THE COMMUNICABLE DISEASES

ALLAN J. McLAUGHLIN

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HARPER'S

PUBLIC HEALTH SERIES

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THE COMMUNICABLE DISEASES

HOW THEY SPREAD AND HOW THEY MAY BE CONTROLLED

BY

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HARPER & BROTHERS, PUBLISHERS
NEW YORK AND LONDON

THE COMMUNICABLE DISEASES

First Edition

K–X

TO
MY WIFE

CONTENTS

PREFACE

THE advancement of science in the past two decades has placed in our hands every necessary weapon for eradicating completely many of the communicable diseases. Why, then, are these diseases allowed to take their toll of thousands of preventable deaths each year?

Because official health departments, unsupported by the voluntary effort of the individual citizen, cannot control these diseases adequately, much less eradicate them.

The control of the "carrier" and of the mild cases which spread these diseases can be secured only by the active, intelligent participation of every citizen in the health movement. The determining factor is the practice of personal hygiene, and the awakening in the citizen of a sense of obligation to the community. Neither of these objectives can be reached by compulsion or official activity based on police power.

The answer to the problem is education, and this book was written in an effort to supply one of the needs of the situation. We have a wealth of technical literature, textbooks on the various specialties of public health. We have, also, a very considerable literature with translations in foreign languages made up of leaflets, pamphlets, and "don't" lists of kindergarten simplicity. This latter is frankly aimed at the lowest stratum, where the need of education is certainly greatest. The highly technical textbooks serve the specialists and technicians. There is a need for books for the use and education of the great intelligent group which comes between the class served by the textbooks and the class which can digest only the simple leaflet.

This highly intelligent group is made up of doctors, lawyers, clergymen, teachers, social workers, clubwomen, business men, business women, and others. They have the

intelligence to read and digest the technical books, but they will not take the time to do it. They object to the loss of time and the mental gymnastics involved in following theoretical discussions without definite conclusions. They desire to have the facts stated in plain language, stripped of academic discussion of theories and hypotheses. Every member of this class is a potential group leader and educator. Each member, if furnished the background and the facts, will, by word and example, educate many of those around him.

There has been much discussion in the past as to the terms, communicable, infectious, and contagious. The word communicable has, in recent years, been in general use to cover all diseases which can be "communicated" either directly or indirectly from one person to another. "Communicable" has a broader significance and includes some diseases that were excluded by the older terms, contagious and infectious. For practical purposes, contagious and infectious are synonymous, and they are used interchangeably throughout the book.

In a work on the prevention of disease, symptoms and treatment need receive but scant attention. They are the business of the practicing physician. Symptoms are mentioned here only as an aid in recognizing the disease, and discussion of treatment is limited to a few diseases, in which the treatment is bound up with the prevention.

The diseases covered are the communicable diseases usually reportable by law to the health department. Tuberculosis and the venereal diseases are problems of such tremendous importance that a proper treatment demands a special book on each. They are, therefore, covered in only a very general way.

In writing this book, I have availed myself of the splendid new book by Vaughan, and the standard works of Chapin, Rosenau, and Park. I have also used freely the publications of officers of the U. S. Public Health Service. To all of these gentlemen, I tender my grateful acknowledgments.

A. J. McL.

PART ONE

INTRODUCTION

THE COMMUNICABLE DISEASES

CHAPTER I

THE CAUSES OF DISEASE AND THE DEVELOPMENT OF BACTERIOLOGY

From time immemorial man has believed that epidemics or plagues were due to supernatural agencies. In the earlier civilizations, it was common to ascribe epidemics to the work of demons or evil spirits. The Jews considered them an exhibition of the wrath of an offended Jehovah and a just chastisement for sin. These two views, that they were due to demoniacal malice or the wrath of God, have come down almost to our own times. Martin Luther leaned toward the demoniacal theory and said that pestilence and disease were the devil's work; while Cotton Mather considered disease as the scourge of God for the sins of the world.

The fascinating theory, that epidemics were caused by the position and movements of the heavenly bodies or other astral phenomena, was often advanced in early times and has its exponents in our own day. Noah Webster believed that earthquakes, volcanic eruptions, and other disturbances, had something to do with epidemics.

Through all the ages, the most consistent theory of the causation of epidemics was that they were due to pestilential air. This theory was advanced by Hippocrates and had

vogue for 2200 years. All the subsequent theorists accepted the bad air theory, but differed among themselves as to whether the bad air was caused by evil spirits, an angry God, juxtaposition of heavenly bodies, terrestrial disturbances, or climatological phenomena.

These theories that foul air or miasmas caused disease were stoutly held and seemed to receive scientific support from the work of the great Bavarian scientist Pettenkofer. Out of these theories were evolved certain fallacies which in the middle of the nineteenth century had wide acceptance. It was believed that disease originated in filth and that miasmas, noxious gases, and foul odors spread disease. This belief fitted in with the very old theory of spontaneous generation, and the two false concepts were destroyed together by the brilliant work of Pasteur.

Much of our knowledge of bacteriology or "germs" is due to the discussions from the earliest times of spontaneous generation and the cause of fermentation. The early Greek philosophers believed that animals generated spontaneously from inanimate matter and these beliefs came down through the Middle Ages. In the sixteenth century, Cardano stated that water gave rise to fish and animals and was also the cause of fermentation. In the seventeenth century, van Helmont gave directions for producing artificial mice, and other writers described the production by water of small animals. The theory that small animals were produced spontaneously began to be disputed in the seventeenth century, and Redi's experiments showed that maggots did not appear in meat, unless flies had access to it and could deposit their eggs upon it.

The microscope was developed in Holland, in the last decade of the sixteenth century. Anthony van Leeuwenhoek

(1632-1723) of Delft, Holland, using a simple lens of a powerful type, was able to see in water and infusions minute living bodies, which we now know were bacteria, and which he called "animalcules." Because these "animalcules" moved themselves about in a remarkably energetic way, van Leeuwenhoek considered them animals, and this opinion prevailed for two centuries. Van Leeuwenhoek's discovery was taken, pretty generally, to mean that these minute living organisms developed "spontaneously" from dead matter.

An Irish monk, Needham, in 1748, attempted to prove spontaneous generation by watching the development of animalcules in infusions, which had been heated in corked flasks, by placing them in hot ashes. Needham claimed that his heating destroyed every living thing in the sealed flasks; and consequently subsequent development of living bodies must be "spontaneous generation" from dead material.

Spallanzani, in 1765, showed that Needham had not sufficiently heated the flasks by hot ashes, and that flasks sealed and heated sufficiently remained clear and free from microorganisms. Spallanzani's demonstrations did not receive universal acceptance, but had one very practical result: they were directly responsible for the work of Appert, who, in 1810, showed that these principles made possible the preservation of meat, fruits, and vegetables; and laid the foundation of the modern canning industry.

The only serious criticism of Spallanzani's experiments was that by heating, the air in the flask was so altered that spontaneous generation could not take place. This criticism was answered by Schultze and Schwann, in 1836-7, who showed that ordinary air carried germs into the flasks, and that air, in which germs had been destroyed by heating or

passing through sulphuric acid, could be admitted without development of germs in the fluid.

In spite of the work of Schwann, Schultze, and others, the scientific world was unconvinced until Pasteur dealt the theory of spontaneous generation the final blow, and definitely fixed in scientific minds that the development of germs, in flasks or in any other situation, was possible only when living germs had been introduced from the air or by other means. The demonstration that germs were in the air was not an unmixed blessing and was responsible for the many futile and erroneous measures of disinfection which had vogue before we learned to differentiate between harmless germs and disease germs, and that the disease germs could not live long outside the body.

In the seventeenth century, Robert Boyle, the great chemist, said that the man who found out all about fermentation would read the riddle of the causation of disease. This remarkable prophecy was fulfilled two hundred years later by Pasteur who, in clearing up the mystery of fermentation, demonstrated the specificity of germs. This was the foundation for all the succeeding bacteriological work which linked special germs with special diseases.

Pasteur showed that fermentation was caused by living organisms, and that each kind of fermentation—milk, wine, or beer—is caused by a special germ. He showed that heating sufficient to kill these germs prevented fermentation. This was the foundation of what we call to-day pasteurization. From Pasteur's work it was a short step to the position, that each disease was caused by a special germ.

The old idea, which prevailed up to Pasteur's time, was that these minute living forms, which moved about in fluids, were little animals because of this movement. Pasteur

described the germs responsible for fermentation as ferments, and considered the motile ones as minute animals.

A very great step forward was the application of the aniline dyes by Weigert to the staining of germs. This staining brought out the structure of germs, and made possible a more careful study and accurate classification. The improvement in the microscope, due largely to the work of Abbé, and the introduction of solid media, gelatin and agar, instead of broth, in which germs were grown at first, were also factors in differentiating germs and fixing their responsibility in the causation of disease.

It is easy and tempting to get into complicated classifications of the single-celled organisms which cause disease; but it is also unnecessary for all practical purpoes to go to any such length. The germs which cause infectious disease may be divided first into bacteria and protozoa. Bacteria are generally considered as belonging to the plant kingdom; and protozoa to the animal kingdom; but dogmatic classification is difficult in living organisms consisting of a single cell which suffices for all their life processes. Bacteria differ from plants and resemble the fungi in being devoid of chlorophyll, the green substance which enables plant cells to absorb and utilize carbon dioxide in light. Many of the bacteria are capable of independent motion in fluids, by means of whiplike attachments, called flagella. These two qualities, motility and absence of chlorophyll, give them a resemblance to single-celled animals—the protozoa.

The lowest forms of animal life consist of a single cell, and are called protozoa. Their structure and their method of reproduction are more complex than the bacteria. It is much better to consider these microbes which cause disease as primitive forms of life, without attempting any definite

classification as plants or animals; to remember that the majority of the known causes of disease are bacteria: but that certain dieases are caused by protozoa.

Filterable Viruses.—There are a number of diseases caused by organisms so minute that they cannot be recognized and classified. These organisms are so small that they readily pass through the pores of a porcelain filter, and hence each of these is called a filterable virus.

Bacteria multiply by fission, which is a division of the single cell into two. In this they differ from the yeasts, which multiply by budding, and the protozoa, unicellular animals, with their more complicated method of reproduction. Bacteria are measured by the micron, which is 1/1000 of a millimeter, or about 1/25000 of an inch. There is great variation in size but the great majority of disease-producing bacteria are from one to five microns long, with a width of less than half the length.

Bacteria are classified according to shape. A spherical bacterium is called a coccus. A coccus which grows in chains is called a streptococcus; in pairs, a diplococcus. A rod-shaped organism is called a bacillus, a curved organism is called a spirillum or a vibrio.

Claims have been made that protozoal parasites cause rabies, smallpox, chickenpox, influenza, and many other diseases of unknown origin. For the present these must be classed as diseases of unknown etiology.

In the following diseases the germ cause is unknown: Influenza, smallpox, typhus fever, dengue, scarlet fever, measles, poliomyelitis (infantile paralysis), German measles, mumps, chickenpox, and Rocky Mountain spotted fever.

The following table gives the principal diseases caused by bacteria.

BACTERIAL DISEASES.

Disease	*Cause*	*Shape*	*Common name*
Tuberculosis.	B. tuberculosis. (a) human. (b) bovine.	bacillus.	tubercle bacillus.
Pneumonia (lobar).	diplococcus pneumoniæ	diplococcus.	pneumococcus.
Leprosy.	B. lepræ.	bacillus.	leprosy bacillus.
Cerebrospinal meningitis.	Diplococcus intracellularis.	diplococcus.	meningococcus.
Diphtheria.	B. diphtheriæ (Klebs-Loffler).	bacillus.	diphtheria bacillus.
Whooping cough.	B. pertussis (Bordet Gengou).	bacillus.	whooping cough bacillus.
Paratyphoid fever.	B. paratyphosus.	bacillus.	paratyphoid bacillus.
Typhoid fever.	B. typhosus.	bacillus.	typhoid bacillus.
Asiatic cholera.	Vibrio choleræ	vibrio.	cholera vibrio.
Dysentery (bacillary).	B. dysenteriæ.	bacillus.	dysentery bacillus.
Tetanus (lockjaw).	B. tetani.	bacillus.	tetanus bacillus.
Glanders.	B. mallei.	bacillus.	glanders bacillus.
Anthrax.	B. anthracis.	bacillus.	anthrax bacillus.
Plague.	B. pestis.	bacillus.	plague bacillus.
Malta fever.	micrococcus millitensis.	coccus.	Malta fever bacillus.
Septic sore throat.	streptococcus hemolyticus.	streptococcus.	hemolytic streptococcus.
Gonorrhœa.	gonococcus (Neisser).	diplococcus.	gonococcus.

The following diseases are caused by protozoa:

DISEASE	CAUSE
Syphilis.	treponema pallida.
Malaria.	plasmodium malariæ.
Amebic dysentery.	entameba histolitica.
Yellow fever.	leptospira icteroides.

Measles and poliomyelitis are caused by a filterable virus, and the organism, which must be very small (ultra-microscopic), has not been identified.

CHAPTER II

THE DEVELOPMENT OF EPIDEMIOLOGY, THE USE OF VITAL STATISTICS, AND THE IMPORTANCE OF CARRIERS IN MILD CASES

Epidemiology is the study of disease, its causes, its manner of spread, and its prevention. It was at first restricted to the study of epidemics. Epidemics always begin with a single case or a group of cases, and the epidemiologists soon found that their most valuable work was done between epidemics, in studying scattered (sporadic) cases. The derivation of the word epidemiology, and the methods of its exponents permit and demand a broadening of the field to a study of all disease epidemic and endemic, noncommunicable as well as communicable. It is derived from Greek words, *"epi"* upon and *"demos"* the people; and it is quite proper to consider epidemiology as a study of all diseases as they affect groups or masses of people. The epidemiologist in practice to-day is quite as much interested in increased prevalence of pellagra or beriberi, as he was formerly in typhoid or diphtheria.

The epidemiologist is the Sherlock Holmes of preventive medicine. He traces the criminal responsible for the outbreak, and fixes the responsibility of his accessories. There have been in all the ages men whose mental make-up would make them epidemiologists to-day. Previous to the nine-

teenth century, these men were acute clinical observers and pathologists, but without exact knowledge of the cause of disease, without facilities for recognizing germs, they were forced to theorize and form hypotheses; and those with the more active imagination spoke of the cause of infectious disease as *"contagium vivum"*—a living virus.

For this vague, indefinite idea, that infectious disease was caused by minute living organisms, Pasteur substituted the definite facts of the specificity of germs: each disease could be caused only by its own specific germ, and this germ always produced the same disease.

With the facts established by Pasteur, Koch, and their successors, the epidemiologist was able to combine the knowledge of the clinical observer, the pathologist, and the bacteriologist. The clinical observer was primarily interested in the sick patient, and he faithfully recorded facts concerning symptoms of the disease. The bacteriologist was primarily interested in the germ which caused the disease, how it lived and reproduced itself. The pathologist was specially interested in the effect of the germs on the tissues of the human body.

The epidemiologist took the information furnished from all three types of observers, and from these data tried to establish how the disease spread, and how it could be checked. His primary interest lay in establishing the modes of transmission, or the chain of infection by which germs traveled from the sick to the well; and in seeking the links in this chain which could be broken by quarantine isolation, disinfection, or such other means then at his disposal with the purpose of preventing the spread of the disease.

The epidemiologist, by his accurate field observation, often points the way to the research worker in diseases whose

cause and manner of spread is unknown. A striking example of this is the classical observations of Carter, of the U. S. Public Health Service, in yellow fever. Carter, in 1898, noted that in yellow fever outbreaks there was a period of about two weeks between the first case of yellow fever and the secondary cases due to the first case. He called this period "extrinsic incubation" or an incubation outside the human body. This observation pointed to insect transmission, and influenced the U. S. Army Commission, which demonstrated the transmission by mosquito two years later.

There was one other source of information which the epidemiologist utilized with tremendous advantage—vital statistics. Vital statistics really form the basis of his work. With knowledge of the cause of the disease, its effect on the patient, and on the patient's tissues, he needs to know only where the cases are in order to break the chain of infection, which links the sick with the healthy. This knowledge is given to him or should be available through vital statistics.

Vital Statistics.—A discussion of vital statistics can be simple. It is possible to strip off the camouflage of higher mathematics, with which some of its zealous devotees have surrounded it. By using complicated formulæ and a terminology made up of big words not in common use, some statisticians restrict the usefulness of their work to the limited few who can understand it. The man or woman, interested in public health work, will read and utilize scientific material in simple language, but usually will not take the trouble to read and grasp the facts, if mental gymnastics are necessary in the process. Vital statistics are so necessary in the prevention of disease that it is not surprising that their use has greatly increased as scientific disease prevention has developed. Vital statistics are a part of the vast fund of

information concerning the nation as a whole, which is collected by the census.

Census enumeration of people is as old as history. Even in the earlier civilization, before the Christian era, some crude attempts were made at census taking for purposes of taxation and military service. Registration of births, marriages, and deaths has been practiced in England for centuries. The collection of vital statistics and their use for the purposes of disease prevention is largely a development of the last fifty years.

The first general census of the United States was made in 1790, and every ten years since that date a census has been taken by the Federal Government. The census collects a great mass of valuable information concerning the social and economic status of our population, but is chiefly valuable to the health official for its statistics of population and mortality. The population statistics form the basis for computing rates of disease and death.

The prevention of epidemic diseases depends upon early knowledge of the existence of cases of disease. The reporting of cases of disease, with births and deaths, furnishes the facts from which vital statistics are compiled. Obviously, for the actual prevention of the spread of disease, the most important fact is the case reporting (morbidity). The reporting of deaths (mortality) is very important and useful in studying the cause and manner of spread of the outbreak, but the report of a death comes after much damage has been done.

Morbidity Statistics.—In 1884, Massachusetts enacted a state law requiring the immediate reporting of smallpox, scarlet fever, and diphtheria, and other diseases dangerous to the public health. This obligation was placed upon house-

holders and physicians. Practically all states now have laws for compulsory reporting of communicable diseases, and the list has grown to large proportions. A model law was adopted by the conference of state and territorial health officers with the U. S. Public Health Service, in 1913; and this has been adopted by many states. The list of reportable diseases is as follows:

Actinomycosis.
Acute infectious conjunctivitis.
Anchylostomiasis (hookworm).
Anthrax.
Cerebrospinal meningitis (epidemic).
Chicken pox.
Cholera.
Dengue.
Diphtheria.
Dysentery (amebic).
Dysentery (bacillary).
Favus.
German measles.
Glanders.
Gonorrhea.
Leprosy.
Malaria.
Measles.
Mumps.
Paratyphoid fever.
Plague.
Pneumonia (acute lobar).
Poliomyelitis.
Rabies.
Rocky Mountain spotted or tick fever.
Scarlet fever.
Septic sore throat.
Smallpox.
Syphilis.
Tetanus.
Trachoma.
Trichinosis.
Tuberculosis (pulmonary).
Tuberculosis (other than pulmonary).
Typhoid fever.
Typhus fever.
Whooping cough.
Yellow fever.

Every ten years we have an official census of the population, and for the years between official censuses we make estimates based upon the rate of growth, as shown by previous census enumerations. No claim for absolute accuracy can be made for such estimates, but they are sufficiently accurate for practical purposes.

General death rates (deaths from all causes) are usually expressed as deaths per thousand persons in the population for one year. Disease death rates are usually expressed as the number of deaths from that particular disease for each 100,000 population. For example, if 5 deaths occurred from typhoid fever in a town of 20,000, the typhoid death rate would be 25 per 100,000. The morbidity rate or the case rate is usually expressed as the number of cases reported of that particular disease per 100,000 population. The fatality rate is the ratio between the number of cases and the number of deaths. It is usually expressed as a percentage: *i. e.* the number of deaths per 100 cases.

Fatality rates and morbidity rates are notoriously inaccurate, because in thousands of mild cases a doctor is not called and they are unrecognized and unreported. There is, also, a diminishing number of doctors who fail to report the cases. This results in some absurd computations where, in some diseases, the deaths outnumber the cases. In spite of incomplete reporting, morbidity statistics are valuable, because every case reported is a focus of infection which can be controlled. This incomplete reporting forces us to place greater reliance on mortality statistics. The report of deaths is more complete, because deaths must be reported in order to secure the burial permit under our laws.

The most serious defect in our machinery for preventing the spread of communicable disease is failure to secure prompt report of cases. Report of deaths is of little value in stopping an epidemic. The reporting of cases by doctors is becoming, year by year, more prompt and more complete. If doctors saw all the cases, it would not be long before we would have a very satisfactory morbidity or case reporting. Unfortunately, two classes of cases described

later, healthy "bacillus carriers" and mild unrecognized cases, are not usually seen by doctors. The only effective way of securing the prompt report of mild cases, "not sick enough to call the doctor," is by the co-operation of the head of the household with the health department. This occurs so rarely that its effect as yet is negligible. Bacillus carriers are also rarely known, and go their way spreading disease, practically uncontrolled.

Carriers.—The most important epidemiologic fact added to our knowledge in recent years is the discovery that healthy individuals carry disease germs, and spread disease without having any symptoms of the disease themselves. These persons are called "carriers." Diphtheria bacilli were found in healthy persons, by Löffler, in 1884. Typhoid fever carriers were first described in England before 1900, but the first emphasis placed on their importance was by Koch, in the campaign against typhoid in southwest Germany. Dunbar, in Hamburg, found healthy carriers of cholera germs, in 1892.

In spite of these earlier findings, the rôle of the carrier was not appreciated until recent years; there was a tendency to regard them as freaks. German investigators, in 1905, demonstrated that the cholera was brought into Germany from Russia by carriers. The writer was working in the laboratories of the Institute for Infectious Diseases in Berlin, at the time of this demonstration (October, 1905, to February, 1906). The general attitude towards carriers, in spite of the German findings, was one of skepticism as to their importance in spreading disease, and the tendency to regard them as medical curiosities was common outside of Germany.

Two years later, 1908, I began investigations in the Philip-

pines to determine what part the carrier played in the spread of cholera. Bilibid prison was an institution under rigid discipline, with absolute control of all food and drink brought in; quarantined against the outside world, with isolation and observation of all incoming prisoners: yet, annually, there was an outbreak of cholera in the institution, which was a perennial mystery to the authorities. An examination of the stools of 264 healthy individuals, waiters, cooks, and other food handlers, showed 17 carried cholera germs in their discharges. Examination of the discharges of incoming prisoners was substituted for the ordinary quarantine and observation; carriers were detected and isolated; and cholera outbreaks in the prison ceased.

A brilliant piece of epidemiologic work, by Soper of New York, did much to impress upon the public and the profession the serious menace of the typhoid carrier. Soper uncovered the damage done by the famous carrier "Typhoid Mary." This woman was a cook and left a trail of typhoid wherever she worked. She worked in seven different families in seven different towns or cities, from 1900 to 1907, and in these families she caused more than twenty cases of typhoid.

Carriers are few in measles and smallpox; but in practically all the other infectious diseases, they play an important part. In poliomyelitis and meningitis, in pneumonia and influenza, they are quite as important as in the diseases in which they were first demonstrated: diphtheria, Asiatic cholera and typhoid fever.

Atypical or Missed Cases.—Next to the carrier in importance in spreading disease is the atypical case, which, because of the mildness of the symptoms or the lack of classical typical symptoms, is unrecognized or missed. The

writer found in the Philippines, that hundreds of cases of children's deaths were reported as meningitis or enteritis, which autopsy showed to be true cholera. A close observation of clinical cases and post-mortem findings showed that there was some excuse for the false diagnosis, as the more severe cases had often meningeal symptoms; and with the exception of diarrhœa, had few, if any of the typical symptoms of cholera. These cases were unrecognized and unreported; and the milder ones, with perhaps only a slight diarrhœa, were able to play around and spread the disease over a wide area.

Diphtheria is frequently so mild and atypical, that its recognition is possible only by laboratory methods. These cases are seldom seen by a doctor and are a big factor in the spread of this disease. In scarlet fever, we are handicapped by not knowing the cause, and consequently cannot call on the laboratory for a positive diagnosis; yet it is equally certain that there are many mild and atypical cases of scarlet fever. Chapin says:

> The layman and the inexperienced physician are apt to scoff at the suggestion of scarlet fever unless the patient has a high fever and is as red as a lobster. But all who have seen much of this disease know that it is exceedingly common to see cases with a scarcely discernible indefinite rash lasting for only a few hours, a rise in temperature of only a degree or two lasting only a few hours also, and the merest trace of sore throat. Sometimes the rash may be entirely absent and even the fever may escape the most careful observation. In institutions and families, such cases, considered doubtful at first, or perhaps entirely neglected, prove to be the origin of typical symptoms in others. Every health officer will recall many such cases. They are the missed cases which are such a factor in the maintenance of this disease.

Some regard measles as a partial exception among the epidemic diseases of childhood, in probably having fewer atypical and mild cases; yet, even here, we must reserve judgment until discovery of the microbic cause gives us the means of laboratory diagnosis, by which mild cases can be positively diagnosed. Practically all the other common infectious diseases have a large percentage of mild or atypical cases, especially poliomyelitis and meningitis.

CHAPTER III

QUARANTINE, ISOLATION, AND DISINFECTION

The three main weapons of the health officer of the nineteenth century were quarantine, isolation, and in the last decade, disinfection. These weapons are still in our hands, greatly modified and more scientifically applied. They are now applied with a more precise knowledge of their usefulness and their limitations. We have passed through an era, in the last decades of the nineteenth century, when each of these measures was believed to be sufficient to "stamp out" an epidemic. This hope was a delusion and the disappointment is compensated for somewhat by the knowledge of why they failed: the demonstration of the importance of the healthy carrier, and the mild unrecognized case.

These terms, quarantine, isolation, and disinfection, overlap somewhat, especially in municipal health work. Many writers speak of quarantine, when they mean isolation of a patient. I prefer to restrict the term quarantine to the detention of an entire household in quarantine, patient plus contacts and suspects. I prefer to use the term isolation in the very modified quarantine, where although the house is placarded, only the patient and one attendant or nurse are isolated, and the other members of the household allowed to go freely. Disinfection had such a definite meaning up to twenty years ago, when we tried to disinfect the air in

rooms, and all sorts of fomites, that I shall describe the very precise disinfection of the patient's discharges and his dishes, and other things used in the sick room, as part of the technique of isolation.

Quarantine.—Quarantine is an exhibition of the natural human instinct of self-preservation. If we look for its earliest application, we must go back to the dawn of history. Repeated instances of what in effect was quarantine practice, are to be found in the Bible and the Mosaic law. Maritime quarantine or the inspection and detention of ships, with treatment of crew and cargo, was first put on a systematic basis by the Venetians.[1] They established a quarantine station on one of the islands, with a delimited anchorage for vessels, detention barracks, and a place where crude disinfection or "purification" was carried out.

Quarantine of vessels from foreign ports was first instituted in America by Massachusetts. In 1700 inspection seems to have been in force and masters of vessels were required to furnish to the selectmen of towns lists of passengers. A little later, the town of Boston established a hospital on Spectacle Island, and, in 1736, an agreement between Boston and the commonwealth provided for a quarantine establishment on Rainsford Island. Later, practically all states and seaports put in effect quarantine laws. These laws and their execution were so diverse in the various seaports, that quarantine practice was chaotic and unduly restrictive of commerce. The unequal application of so many quarantines resulted in the passage by Congress of the Quarantine Act

[1] Quarantine derives its name from the Italian word *quaranta* meaning "forty," from the forty-day detention period required by the Venetians.

of 1893, which provided for uniformity of law and practice in the treatment of vessels by all quarantines under the supervision of the federal government. The interpretation that maritime quarantine was a federal function was so obvious in the law, that since that time the entire quarantine protection system of our seaports has been transferred to the federal government by the states and cities, and is now operated by the United States Public Health Service.

By its maritime quarantine the federal government restricts its activity to the protection of the United States against plague, cholera, smallpox, yellow fever, typhus fever, and leprosy. Quarantine in our times has become modified and humanized; but it is still the clearest example of police power exercised by health authorities.

Interstate Quarantine.—In addition to the police power exercised by the federal government's maritime quarantine in our seaports, the federal government has additional police power under the Constitution and by statute for the prevention of the introduction of communicable disease into one state from another. This police power for the prevention of the spread of disease from one state to another is called interstate quarantine. There is also police power, implied but not expressed in the Constitution, inherent in the federal government in connection with the general welfare and interstate commerce clauses. This power is necessary to cover conditions not amenable to or corrigible by state police power, and its exercise cannot be an usurpation of state authority.

Congress has repeatedly given police power by statute to federal agencies to cover such conditions, but has always maintained the attitude that in health matters the state and

local agencies should be utilized to the limit of their legitimate fields. The Quarantine Law of 1890 gives very definite police powers to the federal health authorities to prevent the introduction of cholera, yellow fever, smallpox, or plague from one state to another, without reference to utilization of state machinery, and provides for the promulgation of rules and regulations with penalties for infraction thereof.

The Quarantine Law of 1893, which includes *all communicable diseases,* provides that the Public Health Service shall co-operate with and aid state and municipal health boards in the execution and enforcement of state laws and regulations and of federal laws and regulations. It provides that where no state or local regulations exist, or where these are insufficient, the Secretary of the Treasury shall make such additional rules and regulations as are necessary to prevent interstate spread of such disease.

It provides, further, that the rules and regulations promulgated by the Secretary shall be enforced by state and local authorities where they will undertake to execute and enforce them; but if state or municipal health authorities fail or refuse to enforce said rules and regulations, the President shall execute and enforce the same and adopt such measures as in his judgment shall be necessary.

In order to carry out this policy of utilizing state and local health machinery in the prevention of the spread of disease, Congress has repeatedly appropriated large sums "to aid state or local boards or otherwise in preventing and suppressing communicable disease" (Epidemic Fund). Congress annually appropriates for the Interstate Quarantine Service, large sums of money for co-operation with state and municipal health authorities in the prevention of the spread of disease in interstate traffic.

The use of police power should be limited to cases in which the result can be obtained in no other way. This conception of the use of police power suggests that it is a necessary foundation, but that, like the foundation of a building, it is something we know exists firm as rock itself, but which we may disregard, and turn our attention to the superstructure built thereon. The interstate quarantine work of the United States Public Health Service is based on such a conception of how police power should be used, and depends for success more upon its opportunity to co-ordinate the efforts of the states than upon quarantining at state boundaries or other police measures.

In achieving national success against any public health problem, the co-ordinative function of the Public Health Service is perhaps the most important function which the service exercises. Some federal co-ordinating agency is necessary, in order to secure a synchronous attack upon any disease with uniformity of method over the entire area of the United States. To secure the maximum of improvement in our national health, we must have nation-wide programs for each problem with which health officers are confronted. The example of our venereal disease campaign serves to show what may be accomplished in other fields by the same methods.

The co-ordinative function of the federal Public Health Service is but the national demonstration of the function exercised by state and local health authorities over smaller areas. In other words, public health organizations —federal, state, and local—should have the following relationship:

Supervisory and Co-ordinating Authority.	*Working Units to be Co-ordinated.*
United States Public Health Service.	State Departments of Health.
State Department of Health.	Local Health Departments.
Local Health Department.	Individual Citizens.

The co-ordinating and supervisory authority furnishes the program in order to secure teamwork and endeavors to have this program carried out, by all the units, in the area within its jurisdiction.

Relation between State and Local Health Departments.—As indicated above, the relation of federal with state health departments has a strong similarity to the relation which should exist between the state and local health departments. The state is the supervisory and co-ordinative authority which makes effective, in a uniform manner, the efforts of the local units. The state should furnish, at least, minimum requirements of a state-wide campaign for each problem, which should be put into effect by the local units. This leaves the local units the opportunity to put into effect any measures in excess of the state's program, which their initiative will suggest and their finances warrant.

In preventing interstate spread of disease, it is the policy of the United States Public Health Service to develop state health departments; and especially those divisions in a state health department, whose effective operation in the interest of the state itself, tends to prevent the spread of disease from one state to another. It is equally manifest that this policy will be futile unless the local health units loyally co-operate with the state health organization. Epidemic disease recognizes no boundaries, local, state or federal. Health

officers to-day recognize that the prevention of communicable disease is never solely a local matter, but that every case is potentially a state and federal matter as well. It is obvious, therefore, that the ideal prevention of disease necessitates a partnership of local, state, and federal health agencies working together, according to a comprehensive nation-wide plan. Obviously, such a plan must contemplate prevention of disease at its source, and must exert its greatest efforts through the local health officer.

State and Local Quarantine.—The state has tremendous quarantine and police power for the protection of the public health, but has delegated this power almost entirely to local health boards. The state still possesses the inherent right (in spite of the delegation of power to local boards) to take such quarantine or other measures as may be necessary to protect its citizens. Even to-day, if a local board of health fails to take prompt and vigorous action, the state occasionally takes charge of the situation, in order to protect the other local units within the state.

The tendency to use police power or compulsory measures is disappearing, and there is a growing tendency among federal, state, and local health executives to secure results by persuasion, by education of communities and individuals. This is logical and sensible, as a strong law may be nullified by an adverse public opinion; while substantial results are frequently secured with a weak law or without law, because of the support of public opinion, expressed as respect for a fine health organization.

While it is true that the prevention of the spread of communicable disease concerns all three official health organizations, it is, nevertheless, in the last analysis, a local problem. It is the local health officer who comes into direct contact

with the individual cases of the disease, and who should apply the measures to prevent its spread to other persons; in fact, a good local health organization, functioning effectively, reduces the necessity for state action to observation, keeping in touch, and lending moral support.

Adequacy of control demands effective action locally, and no activity at the state capital or at Washington can compensate for failure to apply prompt, effective measures on the spot. If the local organization is defective or nonexistent, state or federal aid may supply the temporary organization.

Local or House Quarantine.—Quarantine, theoretically, would stop disease from spreading, but it must be a perfect quarantine. It would of necessity be a non-intercourse quarantine, which is never possible. If all families were completely quarantined for one month in a measles epidemic, measles would disappear; but no such quarantine is possible: business must go on, and people would not submit to such a procedure. Health authorities can quarantine a known case, but healthy families, unless exposed to infection, cannot be restricted in their personal liberty. Modern quarantine has become, of necessity, more of a sieve than a dam.

The use of quarantine locally has been steadily declining, both in the number of diseases which we attempt to control in this way, and in the severity with which it is applied. The discovery that in practically all epidemic diseases there were many carriers and unrecognized mild cases, minimized the results obtained by shutting up in the house a few contacts who had been exposed. The practice to-day is to isolate the patient and one attendant or nurse, and to allow the other inmates considerable liberty, keeping them under observation

for the number of days corresponding to the period of incubation of the disease. The great hardship of quarantining wage earners should be avoided; and it is usually feasible either to isolate the case in the house, with one attendant, or to remove the case to a hospital, allowing the wage earners full liberty.

The police power of quarantine is sometimes used to secure consent to hospitalization, vaccination, or some other procedure in the interest of the public health. The right to impose a quarantine has been upheld by the courts, and as an alternative, consent to hospitalization or vaccination is given in order to avoid the inconvenience of a close quarantine. House quarantine may be reduced to placarding, and this is of decided value. Placarding the house tends to prevent spread of disease from the case quarantined, by giving warning and keeping visitors away.

Isolation.—Isolation should be sufficient in itself to prevent the spread of contagious diseases; theoretically, if all cases could be isolated early enough and completely enough, there would be no spread and the disease would disappear; practically, we know now that all cases mean not only the recognized cases, but the mild or atypical, unrecognized cases, and the carriers who have no symptoms, whatever. We know further that the frank typical cases before isolation are usually allowed freedom for several days, in the most infectious period of the disease. It is true that in some diseases the greatest damage has been done before isolation is effected; still, isolation should be carried out to prevent further damage.

The degree of contagiousness and the length of the period of communicability also influence the value of isolation. In measles, influenza, whooping cough, chickenpox, German

measles, and mumps, diagnosis is not made and isolation is practically never effected until many people have been exposed and most of the damage is done. In scarlet fever and diphtheria, with their long period of communicability, isolation is of undoubted value especially if, in addition to the known cases, contacts and carriers are found and isolated.

Isolation can be carried out best in specially equipped contagious disease hospitals, but it is quite possible in the home of ordinary circumstances and average intelligence to secure fairly satisfactory isolation. Success is contingent upon the intelligence and conscientious care of the attendant in charge of the case. In many families, the lack of facilities and the lack of an intelligent attendant make successful isolation impossible; and hospitalization is the only solution of such a problem.

Chapin did more to put municipal control of contagious disease on a sound basis than any other authority. He pointed out that isolation in specially constructed and equipped hospitals did not prevent the spread of the disease. He, first, brought out, in his convincing way, the limitations of isolation; and very definitely stated that our failure was due to the fact that carriers and mild cases were uncontrolled, and that the cases isolated were not recognized until after several days' freedom, in the most contagious stage of the disease.

Isolation never succeeds in suppressing epidemics of such highly contagious diseases as measles, influenza, or smallpox; but even in these diseases, it prevents many cases, and should be practiced. To fail to isolate any known case of contagious disease in the infectious stage would be the height of folly; but to expect suppression of an epidemic by such means is equally foolish.

Isolation of a patient in a room, without an intelligent nurse or attendant, and without proper facilities for preventing the infective discharges of the patient from leaving the room, is worse than useless. The procedure in the sick room should aim to destroy or disinfect the patient's discharges as soon as possible after their exit from his body.

In practically all the contagious diseases, the discharges from the mouth and nose should be received on gauze or paper; and then can be placed in paper sacks and promptly burned. There should be a tub or large receptacle in the room, in which towels, bed linen, handkerchiefs, etc., can be immersed in a compound cresol two per cent solution or carbolic acid five per cent, formalin ten per cent, or mercury bichloride one to 1000.

Another procedure is to have clean laundry bags in the room to receive the soiled linen; bag and contents to be boiled, immediately, after being taken from the room. Gowns should hang just inside the door of the room, and doctor, nurse or attendant should put on a gown upon entering the room; the gown should be taken off just before leaving the room; and the last act, before leaving the room, should be a thorough disinfection of the hands, in 1 per cent lysol, or other standard disinfectant. Dishes, knives, forks, or spoons should be sterilized in boiling water and should not be taken from the room; no food should be taken from the room; remnants of food should be burned, or treated in the room the same as bowel discharges.

Bowel and urinary discharges should be received in glass or porcelain vessels, containing disinfecting solution. More of the disinfectant should be added, until the disinfectant is twice the volume of the discharges. The disinfectant should be compound cresol solution 2 per cent, carbolic acid

5 per cent, or formalin 10 per cent. Urine and fecal discharges should be allowed to stand in the disinfectant solution for not less than one hour before removal, taking care to break up masses, so that the disinfectant may act upon and destroy all infection.

Disinfection.—With the development of our knowledge of germs, in the last quarter of the nineteenth century, it was natural that disinfection should be a favorite weapon of the health officer. Germs were supposed to be in the air, and to cling to life tenaciously outside the body upon inanimate things (fomites). The laboratory demonstrated that disinfectants would kill germs. It was natural therefore that the health officers of that day should try to disinfect the air of sickrooms, and all sorts of inanimate things which they thought might carry infection. This was the period when letters from yellow fever countries were disinfected; and when gaseous disinfectants such as formaldehyde gas and sulphur had such a vogue.

Gradually, scientific experiment showed that the germs which caused disease are, in the main, frail organisms, and can live but a short time outside the body. In the first decade of this century, we learned to concentrate on man, himself, as the carrier of infection, and largely to disregard fomites, or the carriage of disease germs by inanimate things. We discontinued terminal disinfection and learned to limit gaseous disinfection to sulphur and hydrocyanic acid gas, as fumigants for the destruction of rats and insects. In contagious disease practice two kind of disinfection are distinguished: terminal disinfection and concurrent disinfection.

Terminal Disinfection.—Terminal disinfection was the disinfection of the sick room after death or the removal of the patient. It was formerly insisted upon in every case,

that formaldehyde or sulphur gas be used. Chapin's classical work made all health boards see that it was unnecessary and an unjustifiable waste of money. Careful experiments showed that the methods used were ineffective, and terminal disinfection with gas is no longer used. In place of gaseous disinfection, the sick room now receives a thorough mechanical cleansing with soap and water or bichloride of mercury solution, or other good disinfectant. If concurrent disinfection has been faithfully carried out while the patient was in the room, these measures are sufficient after the removal of the patient: mechanical cleansing, with thorough airing and admission of the maximum of sunlight.

Concurrent Disinfection.—Concurrent disinfection is the destruction of the disease germs in the discharges of the patient, immediately after their exit from the body; the immediate disinfection in the sick room of all things soiled by the patient or which have come in contact with him, including the hands of nurse or attendant. These measures, which constitute the technique of the sick room, have been described under isolation.

CHAPTER IV

INFECTION AND IMMUNITY

Infectious disease is the result of the invasion of the body by germs, and the production within the body by the germs of substances, which are either poisons or capable of being changed by ferments into poisons.

Immunity to disease is that state of the human body, in which disease germs and their products present in the body cause no symptoms. The word resistance is used to denote the same condition of the body, as immunity means ability "to resist" infection by disease germs. The opposite condition to immunity is designated as susceptibility.

Immunity of individuals to certain diseases was noted by the ancients, but the first definite scientific demonstration of immunity was that made by Jenner, who showed that inoculation with cowpox rendered the subject immune to smallpox. Jenner's work was not followed by extension of the great principle of immunity to other diseases until Pasteur's work, about one hundred years later. Pasteur used living germs "attenuated" by heat, by passage through animals, or by other means. The next advance was the discovery that living germs were not essential, and that killed cultures (germs killed by heating) could be used to produce immunity. Killed cultures were first used to produce vaccines against cholera and typhoid fever.

After demonstration of immunity produced by vaccines and general acceptance of the fact, there was and is still no clear conception of the mechanism of immunity. Each decade since 1880, new discoveries have increased our knowledge, and shown that the subject is much more complex than at first supposed.

The explanation was offered by Metschnikoff, that immunity depended upon the activity of the white blood cells or leucocytes. He showed that the leucocytes in the blood and tissues possessed the property of taking up germs and digesting them. This phenomenon of phagocytosis explained how germs could be destroyed. The discovery of antitoxin in the blood serum made the cellular theory inadequate as an explanation of antitoxic immunity.

The next advance was the discovery that the activity of the leucocytes against germs (phagocytosis) was due to the stimulus of substances in the blood serum (opsonins), and that in the destruction of bacteria, two substances (amboceptor and complement) were necessary, either of which was powerless without the other. It is now clear that when a person is immune, he possesses what we call antibodies in his blood, which protect him against the germs and their products.

Antibodies can be demonstrated only by the effects they produce. We know nothing of their chemical composition, as they have not been isolated. They give such definite results, that we know they are in the blood serum or tissues, although we cannot isolate and describe them. Antibodies are produced in the blood or tissue by the inoculation of protein substances, including bacteria, and any substance which causes the production of antibodies is called an antigen. Although we cannot see the antibodies, we know

many of their properties; they are destroyed by moderate heating, and affected by light and chemicals. The antibodies, of various kinds, are produced by the inoculation of an animal with germs or toxins.

Agglutinins are antibodies, which have the property of causing bacteria to "clump" or agglutinate in masses; they are of great value in diagnosis, but of no value in protection against the germs. The presence of agglutinins does not necessarily mean immunity; although the production of agglutinins usually goes hand in hand with the production of the other antibodies, upon which immunity depends. There is present in normal serum a substance called complement, which alone has no effect on an antigen, but combined with an antibody produced by that antigen, the complement becomes active and destroys the antigen.

Lysins are antibodies which dissolve cells, by means of the complement normally in the blood. Antibodies which act by linking the antigen (germ or cell) to the complement, and so permitting the complement to act, are called amboceptors.

Opsonins or tropins are antibodies produced by inoculation of germs (antigen) which have the property of so changing the germs, that they are easily taken up and digested by the leucocytes.

Precipitins are substances produced by the inoculation of a soluble antigen. If some of the antigen is added to the serum of an animal containing precipitins, a flocculent precipitate is produced. When a soluble toxin is used as an antigen, another antibody is produced; an antitoxin which combines directly with the toxin, and neutralizes it.

Ehrlich evolved his famous "side chain" theory to explain the formation of these various types of antibodies. He first

presented the theory as an explanation of how the body cell absorbed food necessary in its life process. His conception of the cell was a central nucleus, with many side chains, called receptors, with affinities for various substances. The bacterial products or toxins may find receptors which link them to the cell, and permit them to damage or destroy the cell. If the cell is not destroyed, new receptors are formed in great numbers: in other words, there is an overproduction of receptors; the injured receptors are more than replaced from the nucleus of the cell. These excess receptors thrown off into the blood serum are the antibodies; attached to the cell, the receptors were the means of damaging the cell; but thrown off from the cell into the blood, they become the protective free antibodies.

Natural Immunity.—There are various kinds of immunity. Natural immunity is an inherited quality by which certain species are immune to diseases common in another species of animal. All the lower animals are immune to typhoid, cholera, yellow fever, measles, syphilis, gonorrhœa, leprosy, typhus fever, and scarlet fever. Man is naturally immune to diseases such as rinderpest or Texas cattle fever. There are certain diseases, such as plague, glanders, rabies, tetanus, Malta fever, and tuberculosis, which are common to man and some animals.

Besides the congenital type of natural immunity, there seems to be a natural immunity developing progressively with age in certain diseases, not dependent upon a previous attack of the disease. This is questioned by many observers, who believe, that in most of these cases, the immunity is acquired by a mild, unrecognized attack of the disease in youth.

There are certain additional factors involved in what we

call natural immunity or resistance to disease, other than the inherent (congenital) protection possessed by the individuals: (1) Mechanical protection by the body tissues and fluids. (2) Dosage of germs. (3) Lowering of resistance by fatigue, chilling, physical depression, or nutritional disturbances.

The skin and mucous membrane, if unbroken and uninjured, offer sturdy resistance to the entrance of germs; but this resistance is purely mechanical in character. The body fluids, secretions of the mouth and nose, the gastric juice, and others have strong bactericidal qualities.

The dosage of germs is important; as a demonstration it may be cited that the animals naturally immune to diseases may be infected by using enormous doses of germs. A guinea pig, immune to cholera germs introduced into its stomach by reason of the acid gastric juice, becomes susceptible if the gastric juice is neutralized by the administration of an alkali, previous to introducing the cholera germs.

The natural immunity possessed by animals may be overcome by larger doses of germs, especially if the factors of chilling of the body, great fatigue, or insufficient diet are added. This point has a practical bearing on human infection, and points clearly to the importance of proper rest, food, clothing, and room temperature, in preventing infection.

Acquired Immunity.—Acquired immunity is the resistance to infection, acquired by having passed through an attack of the disease, or produced artificially in the body by the injection of serum, toxin, vaccine, or virus. Lasting immunity is conveyed by an attack of any of the following diseases: Measles, typhoid, cholera, plague, smallpox, chickenpox, yellow fever, typhus fever, scarlet fever, poliomyelitis,

mumps, syphilis, and diphtheria. An attack of diphtheria in general protects; second attacks occur in less than 1 per cent of cases. No lasting immunity follows an attack of the following: Tuberculosis, pneumonia, influenza, gonorrhœa, pus germs (cocci), dengue, tetanus, erysipelas.

Acquired immunity may be either active or passive. Active immunity is the production in the body of protective substances (antibodies), in response to an attack by disease germs or following the injection of a virus, vaccine, or toxin (antigen). Passive immunity is the temporary protection given a human individual by injecting into his body ready-made antibodies manufactured in the body of an animal, such as the horse. As an example, in diphtheria antitoxin, the toxin is injected into the horse, and the antitoxin is produced in the horse's blood. The horse serum containing the antitoxin (antibodies) is then available tc furnish immediate, though temporary, immunity, by injection into the human body.

Passive immunity is practically limited to combating toxins; germs like diphtheria and tetanus, which produce a true toxin, or the toxins of snake venom, make ideal antigens for producing the antitoxins in animals, which have value in the passive immunity of man. In the bacterial infections, due to germs which during their life produce no true toxin, the immunity is not antitoxic, but depends upon other properties or antibodies in the blood, which bring about the destruction of the germs. For these reasons, passive immunization has not been a practical success, except in the diseases due to a true toxin.

The production of active immunity artificially has a wider field of usefulness. By a harmless injection of vaccines, an immunity equivalent to, or greater than, that acquired

by an attack of the disease is possible now, in a constantly growing list of diseases. As a matter of fact, both forms of immunization, active and passive, have their place in some diseases, as in diphtheria. The active immunity, though lasting, is slow in developing and takes weeks or months to appear; while the passive immunity, though temporary in duration, gives immediate protection (antitoxin).

An interesting fact concerning active immunity is that, while it is a great boon to the individual protected, it permits him to be a germ "carrier" without symptoms, who may cause great damage by spreading the disease.

Latency of infection is also a phenomenon of relative immunity. Most immunity is relative, or it may be stated that absolute immunity is rare. Many cases may be cited where a latent infection develops, for example, when a carrier of germs with sufficient immunity to show no symptoms, suddenly becomes ill, after some experience which lowers his resistance, as fatigue, exposure to cold, insufficient food, etc.

CHAPTER V

SOURCES AND ROUTES OF INFECTION

From the remote ages, man has sought to fix the blame for infection upon other sources than himself. Filth and miasms were blamed, and we have seen that the discovery of germs at first seemed to support the case against filth. It would be a waste of time to review the steps by which we have arrived at our present position. To-day the mists of superstition have been dissipated, and we know that the source of disease germs is man, himself; with the exception of a few diseases with animal sources.

For all practical purposes, the sanitarian may disregard all sources except man, himself, for all the communicable diseases except the following:

DISEASE	SOURCE
Plague	rat.
Rabies	dog.
Glanders	horse.
Malta fever	goats.
Anthrax	cattle.
Tuberculosis	cattle (in part).
Rocky Mountain spotted fever	squirrels, and other small animals.

Experimentally, some of the other diseases may be produced in animals, but animals under natural conditions play

no part, except in the diseases named above. Man is the reservoir of infection, and the greatest progress of the past decade has been in concentrating attention on man, the source.

Routes of Infection.—By what routes do the germs in the body discharges of one person reach the mouth or nose of another?

Direct Infection.—In the diseases which are spread by the discharges from the mouth and nose, these discharges may be transferred from sick to well directly, as by kissing or by "droplet" infection. Where the sick and well are in close contact, the germs are sprayed from the sick in "droplets" into the air, by sneezing, coughing, or even talking; and may be breathed in by the healthy persons, who are close by. These mouth and nose discharges are often transferred, also, by means of the hands. Direct transmission of the bowel and urinary discharges usually takes place by means of soiled hands. All the body discharges may be transmitted from sick to well by drinking cups, dishes, spoons, toys, and a multitude of objects recently soiled.

The difficulty of preventing disease spread from man to man, more or less directly, is forcibly stated by Rosenau:

> The knowledge that most infections are spread rather directly from man to man brings in all the forces of sociology to that of preventive medicine. The task of preventive medicine is thereby rendered much more difficult from the fact that most infections depend upon the control of man himself. We ruthlessly wage war against insects or against infected food or water. In other words, we can arbitrarily control our environment to a very great extent, but the control of man himself requires the consent of the governed. Thus it is easier to stamp out yellow fever than to control typhoid fever. It is easier to suppress malaria than syphilis, rabies than influenza, trichinosis than measles. Cattle

appear to be mutely thankful when protected by inoculation against blackleg or anthrax, but man rebels against one of the best of all specifics—vaccination against smallpox. The fact that man is the chief source and reservoir of most of his own infections adds greatly to the scope and difficulties of public health work and often makes the prevention of disease depend upon social changes. In this sense preventive medicine is a very important factor in sociology.

These various routes of more or less direct infection are usually spoken of as contact infection.

Indirect Infection.—Infection may be carried from the sick to the well indirectly, by means of water, milk, food, soil, and insects. Water very frequently is the medium by which typhoid fever, Asiatic cholera, and dysentery are transmitted. That human sewage reaches water supplies, can be demonstrated; the *bacillus coli* is a harmless inhabitant of the human or animal intestine, and when found in water, shows that human or animal sewage has found its way into the water.

The colon bacillus is accepted as an index of sewage pollution of a water supply, because of the difficulty of recovering the typhoid bacillus. We do not wait actually to demonstrate typhoid bacilli in water; but the potential danger of a supply is shown by the presence of the colon bacillus, and steps are then taken to nullify or remove the pollution.

Water has been accused of carrying many infectious diseases, but responsibility for causing disease may be limited, for all practical purposes, to those diseases in which the exit of germs from the body is through the intestinal or urinary discharges. These germs do not multiply in water and their life therein is relatively short, so that recent pollution by

human sewage may be considered necessary to make a water supply a disease carrier.

Besides cholera, typhoid, paratyphoid, and dysentery, water polluted with sewage causes diarrhœas and infections due to the eggs or larvæ of worms or parasites, which are discharged with human feces.

Milk is responsible for transmitting a longer list of diseases than water. It is such a good culture medium for germs, that they not only live but increase in it; something they are incapable of doing in water. Milk transmits the diseases due to the intestinal discharges, and in addition the germs of many of the so-called "sputum-borne" diseases, or those diseases caused by discharges from the human mouth and nose. Therefore milk may carry, in addition to typhoid fever, cholera, and dysentery, the germs of tuberculosis, scarlet fever, diphtheria, septic sore throat, Malta fever (goat's milk), and other diseases. The milk is infected by human carriers of the disease germs in nearly all instances. There are some exceptions: sewage polluted water used to wash cans or to dilute the milk, can infect the milk; a tuberculous cow transmits the germs with its milk; and Malta fever, a disease of goats, is transmitted by the goat's milk. Ice cream may be infected and carry the same disease germs as milk.

Meat has been accused of carrying disease, and under certain conditions may do so. The fact that it is usually thoroughly cooked nullifies much of the danger; although trichinosis and tapeworms may be transmitted by underdone meat. Most outbreaks of meat poisoning are due to paratyphoid bacilli, or other members of the colon group in the meat, which is eaten raw or underdone; or which, if thoroughly cooked, was infected after cooking, by the hands of

a carrier of these intestinal germs. Botulism or sausage poisoning is due to the growth in sausage of a bacillus *(B. botulinus),* which produces a very powerful poison or toxin. This toxin is destroyed by heat, so here again the damage is due to insufficient cooking. Vegetables, such as beans and spinach, canned with insufficient heating, may have been infected with *B. botulinus,* which produces its toxin in the can, and causes deaths, if the subsequent cooking is not thorough. A number of deaths, due to ripe olives from the same cause, were reported; the olives being heated very little and the toxin in the can being undestroyed. Any food which is thoroughly cooked and not infected by dirty hands, flies, or other agencies, is safe, even if it has contained germs before cooking. Foods eaten raw, oysters, clams, etc., are potentially dangerous; they may be from grossly infected sources, or may be infected in handling or serving.

The soil may furnish a means of indirect infection of man, because it is constantly infected by the discharges of man and animals. It has wonderful powers of purifying itself by means of the harmless (nitrifying) bacteria resident in the upper layers of soil; but certain bacteria, which are capable of causing disease in man, are able to maintain themselves indefinitely in soil. They are usually bacteria which form spores, and have greatly increased resistance for this reason. The principal diseases which man receives from soil infections are tetanus (lockjaw), anthrax, gas gangrene (Welch's gas bacillus), and malignant œdema. These germs do not grow or multiply in the soil, but their spores are not destroyed; and when these reach a favorable medium, as the body tissues of man, they develop and cause disease.

Insect Transmission.—One of the most interesting de-

velopments of preventive medicine is the increasing number of diseases which in the past twenty years have been shown to be transmitted by insects. The insect may act in one of two ways: he may mechanically pick up infection from one patient and carry it to another without change in the germs (bacterial infections), or the germs may undergo reproductive changes in the insect (protozoal infections). There are certain protozoal infections in which the parasites are capable of reproduction in two ways: a sexual reproduction, and a nonsexual reproduction. The insect in these diseases is the true host of the parasite, and the sexual cycle takes place in the insect; when transmitted to man, the parasite reproduces itself nonsexually, more like reproduction changes in ordinary body cells.

As distinguished from the mechanical carriage of bacteria by insects, this transmission of protozoa, which undergo reproductive changes or a life cycle in the insect, may be called biological. Mosquitoes are responsible for the transmission of malaria, yellow fever, dengue, and filariasis. Ticks carry Rocky Mountain spotted fever, relapsing fever of W. Africa, and Texas cattle fever. Bedbugs carry European relapsing fever, and Indian *kala azar*. Lice transmit typhus fever, and some forms of relapsing fever. Fleas are responsible for the transmission of plague. Biting flies transmit small eel-like parasites, called trypanosomes, which cause sleeping sickness and various animal diseases. Flies and other insects can carry mechanically the germs of many diseases, typhoid, cholera, eye diseases, smallpox, and others.

CHAPTER VI

OUR INADEQUATE CONTROL, AND HOW ADEQUATE CONTROL MAY BE SECURED

In the preceding chapters I have outlined the development of bacteriology and our constantly growing knowledge of the germ causes of disease.

High hopes for the eradication of disease were based upon the facts of bacteriology as they rapidly developed from the sound foundation established by Pasteur and his successors in the closing decades of the nineteenth century. Why these hopes of success were not realized is discussed under Epidemiology, Quarantine Isolation, and Disinfection. These hopes were sound enough, for they were based upon the isolation of all cases of disease and the disinfection of the patient's discharges at the bedside.

These measures could be enforced by the police powers of boards of health, and it seemed that vigorous, fearless exercise of this official function would "stamp out" epidemics and eradicate disease. This policy failed and the hopes were not realized because all cases could not be brought under control. It was demonstrated that carriers and mild cases entirely uncontrolled were in many diseases as numerous or even more numerous than the easily recognizable cases with typical symptoms. There was a tendency at first to blame the failure to control all cases upon the doctors

who did not report all their cases, and reported many of them after the greatest damage had been done.

The facts disclosed that a large percentage and, in some diseases, a majority of the cases were untreated by doctors. The cases either had no symptoms (carriers), or they were so mild that a doctor was not called. It was also shown that doctors are commonly called late, after the patient has been exposing others to infection for several days.

These basic facts explain the limitations of strictly official activity in attempting to control communicable diseases. It was obvious that a new alignment of public health forces was necessary, including an enlistment of private citizens in the public health army. If a large percentage of cases, including mild cases and carriers, do not come under official control, or the private control of doctors, we can secure control of these only through the participation of the private citizen in the work of suppressing disease.

Official action in securing safe water supplies, proper sewage disposal, and pasteurization of milk has accomplished spectacular results in typhoid fever and allied diseases.

In the communicable diseases of childhood, the police powers of quarantine, isolation, and disinfection have been utilized to good advantage. They have prevented many cases of disease, but they have failed to eradicate disease. The exercise of these powers is necessary; they should be continued, but health officers must secure support for their official activity from private, voluntary, unofficial agencies and private citizens, if the elimination of many diseases or even further significant reduction of these diseases is to be achieved. Our failure to control adequately or to eradicate many communicable diseases is not due to lack of knowledge,

but to our inability to secure the application of the knowledge we already possess.

We have sufficient knowledge and possess every necessary weapon to combat and eradicate completely typhoid fever, diphtheria, smallpox, syphilis, malaria, and other diseases. We cannot secure the application of this knowledge by official activity unsupported by private agencies and private citizens.

We freely confess that we need more scientific knowledge concerning the cause and modes of transmission of many diseases, but it is patent to everybody that what we need more is a general application by the people of the knowledge we already possess. There is a group of communicable diseases which always arouses public interest and in which there is no difficulty in securing prompt and effective application of our scientific knowledge.

The prevention of this group constitutes one of the major functions of our federal Public Health Service. These diseases are more or less exotic and inspire terror in the lay mind out of all proportion to their danger. In this group are Asiatic cholera, plague, yellow fever, typhus fever, and leprosy. We have sufficient knowledge to control all of these diseases and the terror they inspire insures the support of public opinion and a feverish activity on the part of lay officials and private citizens to effect their suppression. This does not mean that there is no need for further research in these diseases, but simply means that our present knowledge backed by public opinion makes possible what may be termed adequate control.

With the diseases which are endemic in the United States, there is quite a different story. They are of common, every-

day occurrence, inspire no terror, and public opinion is apathetic toward measures for their suppression.

The time has come for health officers to be statesmen rather than policemen. Our people for so many decades regarded the work of our health departments as an exhibition of police power. They were so accustomed to the mandatory orders to do this or not to do that, that they still confine their co-operation to doing enough of what they are told to do, to keep them out of court. They are prone to look upon the advice given to-day by our up-to-date health departments as something unnecessary, because it carries with it no threat of fine or imprisonment.

To secure administration of vaccines we must have something besides compulsory laws. The wide use of these preventive inoculations can be secured only by the voluntary act of the head of the family. Our failure to acquire control of mild cases and carriers, and our failure to secure wide use of prophylactics and vaccines are questions which require an answer, and the answer is obvious. We find in these questions the utter futility of unsupported official action. We know that the answer to these questions involves something personal, the habits of the individual, and the habits of the family group, so that we have reached a point where we know what must be done, but this is something we cannot order done, something that the citizen can be requested to do, but cannot be compelled to do.

Imagine the possibilities in prevention of disease, if parents voluntarily would isolate children and report to the health department, pending diagnosis, in all cases with indefinite symptoms presenting a sudden deviation from normal health. Imagine further the effect upon death rates if every individual would practice personal hygiene to the

extent that his hands were always clean when handling food, and that the discharges from mouth and nose were always properly controlled.

How can we create in the individual citizen, the head of a family, the desire to do his part in personal and community hygiene? How can we secure the actual participation of that family in a joint voluntary effort for the protection of the community? The answer seems obvious: Education. Yet the problem of educating all individuals in personal, family, and community hygiene is of such magnitude that it appalls even the most optimistic.

The schools furnish the most hopeful avenue of approach in educating the individual in personal hygiene and in community obligations. Organization of the children themselves into health leagues is in many communities the only effective means of reaching the parents and changing the unhygienic habits of the home. In other communities, the parent-teachers' associations will be found a most potent factor.

The utilization of the schools is complicated by the fact that there are two jurisdictions and sometimes three involved: the board of education, the board of health, and sometimes a parochial school system. This complication is more apparent than real, and fades away before the health officer who is a real leader. It is really immaterial whether the work to be done comes under the health board or the school board; the vital necessary thing is to have it done. This teaching of personal hygiene must be begun in the early grades because of the fact that 90 per cent of the children do not enter high school, and a very large percentage do not get beyond the sixth grade.

To acquire control over carriers and mild cases, in addi-

tion to the education of individuals in personal and family hygiene, it will be necessary to develop a strong community spirit. This is easier in small towns where it is quite possible, if the health officer has the qualifications for leadership, to secure the voluntary co-operation of a great majority of the householders. The problem in a large city is much more complex and difficult. To secure similar results in large cities requires decentralization of the health department into health units or health centers, which can be used to develop a community spirit in health matters among the majority of the citizens in the area served by the health center.

Careful study must be made in fixing the boundaries of districts served by health centers, in order to secure some degree of homogeneity and cohesion among the individual families served. The limits of the health center's activity need not coincide with those of ward or precinct, but should be made to include a unit not too large whose population is of such a character that it can be bound together in a community spirit. Many excellent societies exist in connection with churches and church work. Unco-ordinated, their activity does not result in community spirit, but in a disintegration of community spirit into little church groups. Their work can be co-ordinated by a committee with representatives of each church group and, under the leadership of the health officer, their combined support can be secured for community health problems. The surest and most effective way of developing community spirit, and securing the participation in disease prevention by large numbers of citizens, is to utilize the many splendid voluntary unofficial associations or societies already engaged in health work and anxious to help.

The greatest single defect in municipal health organization to-day is the lack of machinery for co-ordinating and utilizing voluntary and unofficial agencies in an official plan to insure team work. There has not been too much activity by voluntary and unofficial organizations, but there has been too little utilization and co-ordination of these agencies in a comprehensive plan having for its object the prevention of waste effort, duplication, and conflict. To secure co-ordination of these agencies some form of central advisory committee is essential, and in the large cities the health center, with, perhaps, a subcommittee, will be found to be the most useful instrument for decentralizing and applying the principles of team work locally. Municipal health organization must, therefore, as its primary function, devise means of utilizing all existing voluntary unofficial agencies as an integral part of the official health machine, and recommend the changes in health department organization which are necessary to effect this result.

The term "health center" has been very loosely used as a name for everything from a milk station to a miniature health department. A real health center should be a complete health department. In a small city the health department should be the health center. In larger cities health centers should be established for the purpose of decentralizing official health activity and linking with it every agency carrying on public health activities within the area. It should also serve as a common headquarters, in order to effect the closest co-operation with workers for sociologic and economic betterment. To operate successfully, the health center must have official status, and in addition to the diagnostic and dispensary facilities, the public health nurses, and other official personnel, it should house the liaison

officers, when necessary or advisable, from unofficial or voluntary agencies.

Unofficial voluntary associations are often the result of spontaneous popular movements for the promotion of health. They give the health department the opportunity of reaching the people through groups rather than by the laborious procedure of teaching individuals. All new expansions in public health are best promoted, in their incipiency, by unofficial agencies. After the new departure has a demonstrated value, official funds can be secured and the work taken over by the official health department. Unofficial or voluntary health organizations have been always the pioneers in new health movements. They have initiated many procedures which should have been started by the official health department, and they have operated clinics, public health nursing service, and educational machinery for years before the official health officer could obtain funds for such a project.

In a somewhat long and varied experience, I have never known an unofficial voluntary health agency which was unwilling to accept the supervision of the duly constituted official health authority. I have always found the unofficial health agencies willing to turn over to the official health department any health activity which might be termed official, whenever the health officer was able and willing to assume the responsibility.

The evolution of public health work from the prevention of contagious diseases to the prevention of all disease and further from the negative prevention of disease to the positive appeal for health, has resulted in a very complex health organization. It has brought into the public health field besides the doctor and the sanitary engineer, the public

health nurse, the teacher of physical education, the social worker, and other health workers. This expansion and this complexity make it doubly essential that the unofficial agencies be co-ordinated and utilized by the official health departments. Health departments would find it impossible to secure official funds to do the work now done by unofficial agencies. It is also a debatable question as to how much of this work should be taken over, and how much might better be left to the private agencies. The important thing is to have the work continued under official supervision, dovetailed into the official program, parts of it to be taken over by the official health department when expedient and when official funds are available.

The American Public Health Association, the great representative public health organization of the country, has done much to bring about closer relation between official and unofficial health workers. Its membership includes health officers, federal, state, and local, and every type of health worker—doctors, sanitary engineers, bacteriologists, and public health nurses—from both official and unofficial organizations.

The most hopeful feature of public health development of recent years has been the better understanding between official and unofficial health agencies and the spirit of helpfulness displayed by both in their common crusade for better health. There is now a National Health Council, which combines representatives of all the great unofficial national health agencies, with representatives of the United States Public Health Service and of the state departments of health. There are also, in many states, state public health associations which offer opportunity for team work between official and unofficial activity. Even more important, in our

large cities there is a constantly growing number of health leagues, health centers, or other organizations which under official supervision secure the co-ordinated effort for health of all the official and unofficial organizations in the area.

PART TWO

DISEASES SPREAD BY DISCHARGES OF THE MOUTH AND NOSE

CHAPTER VII

PNEUMONIA

Definition.—Pneumonia is not a clean-cut entity like diphtheria or measles, caused by one specific germ, but under this name are grouped acute inflammations of the lung tissue, caused by a dozen different germs. The division of pneumonia into lobar pneumonia and broncho-pneumonia is somewhat logical, and the differentiation is in most cases easily made; yet there are cases which seem to combine both forms.

Acute lobar pneumonia is an acute, febrile, infectious disease, with a strong predilection for young adults, beginning abruptly as a primary pneumonia, in which one or more lobes are included, the air spaces and tubes filled with a fibrinous exudate.

Broncho-pneumonia is seldom primary, but is usually secondary to measles, influenza, whooping-cough, and other diseases. It is the common pneumonia of childhood and old age. It affects smaller areas, lobules instead of lobes; and there may be patches all over the lungs, without consolidation of an entire lobe. The exudate into the lobules and bronchi is catarrhal, made up of mucus and pus, not fibrinous as in lobar pneumonia.

Ninety per cent of lobar pneumonia is caused by the pneumococcus, and there are various types of pneumococci

Broncho-pneumonia is most often caused by the *streptococcus hemolyticus,* but several other germs can cause it.

It is obvious that the classification of the pneumonias is unsatisfactory, and for purposes of prevention it is not material. The same measures are necessary and effective in preventing all types of pneumonia. We must think of pneumonia as being caused by many different germs; and that all of these germs, passing through the trachea to the lungs, are capable of growing and multiplying, setting up an inflammation which constitutes a pneumonia, either lobar or lobular.

History.—The earlier medical writers knew and described pneumonia. In the fifteenth and sixteenth centuries, it was recognized as a contagious disease. Toward the end of the eighteenth century, the consolidation of the lung to the consistence of liver was noted, for which the name hepatization was coined. Noah Webster described a "putrid pleurisy" in Waterbury, in 1712, and refers to it as a disease which often made dreadful havoc in America. It has always been an important factor in the mortality of armies. In the Civil War, the losses from pneumonia were heavy, but were obscured by the greater prevalence of the intestinal diseases, dysentery and typhoid. During the World War, advances in science made possible the reduction of mortality from typhoid and dysentery to a negligible quantity, so that the mortality from pneumonia stood easily first, as a cause of death. In the civilian population, pneumonia kills more people than any other single cause, except heart disease. It is responsible in the United States for over 10 per cent of all deaths.

Acute Lobar Pneumonia.—Acute lobar pneumonia is pre-eminently a disease of the strong and robust, between

the ages of 15 and 50 years. It usually begins suddenly with a chill, followed by high fever, great prostration, and difficult and painful respiration. After about seven days, there is an abrupt change or crisis, with either a fatal termination or a sudden betterment of all symptoms, and a quick recovery.

The cause of lobar pneumonia is a small lancet-shaped coccus, usually in pairs, hence called *diplococcus pneumoniæ*. The common name of the organism is the pneumococcus. It is found in the mouths of healthy persons and in the dust of rooms occupied by pneumonia patients. It was formerly believed that all pneumococci were the same, but now we know that there are at least four distinct types. This differentiation of the members of the pneumococcus group causes us to modify our views as to the significance of pneumococci found in healthy persons. The fact that pneumococci were found in the mouths of healthy persons, caused many to believe that the chief factor in infection was the resistance of the individual. It seemed that the pneumococcus was present, waiting for some other factor to lower resistance and permit infection of the lungs to take place.

The work of Dochez, Avery, Cole and others, has established four types, with several subdivisions. In 454 cases at the Rockefeller Institute, 33 per cent were due to type I, 33 per cent to type II, 12 per cent to type III, and 20 per cent to type IV.

Immunity.—There is usually said to be no immunity to pneumonia; that one attack does not protect against another. In all probability, an attack due to type I produces an immunity to type I of short duration, probably several months; but would have no immunity against types II, III, or IV. Thus the discovery was made that the types responsible for

about 66 per cent of lobar pneumonia were not found except in cases of pneumonia, in the dust of the pneumonia sick-room, and in the mouths of a few carriers who had been in contact with pneumonia cases. These facts establish acute lobar pneumonia as an acute communicable disease, which should be treated as diphtheria is treated, in order to prevent its spread.

It has been demonstrated, that about two-thirds of all lobar pneumonia is due to types I and II; and that these organisms are rarely found in the saliva of healthy persons. About 81 per cent of pneumococci found in the saliva of healthy people are found to be types III and IV. Type IV is most common in the saliva of healthy persons, yet it causes only 20 per cent of pneumonia cases, and these are usually mild in character and seldom fatal.

Cole developed a serum which has a curative value against a type I infection only. In type I infections it seems to reduce the mortality from 30 per cent or higher to 8 or 10 per cent.

Vaccines (killed cultures) have shown promising results. Cecil and Vaughan vaccinated 80 per cent (13,460 men) at Camp Wheeler. Of the vaccinated only one in 1,680 contracted pneumonia; while among the unvaccinated, one in 80 came down with the disease.

Broncho-pneumonia.—Broncho-pneumonia or lobular pneumonia is the common secondary pneumonia, which follows measles, whooping cough, influenza, and other diseases. It is most commonly caused by the streptococcus, but may be caused by the pneumococcus, the Pfeiffer bacillus or the bacilli of plague, anthrax, typhoid, and other germs. The streptococcus, as its name implies, is a minute spherical organism, growing in chains. There are probably many

groups or types of streptococci. They are constantly present in the skin and mucous surfaces of the human body, and in the soil, water, and milk. They are present in strategic positions to invade the body, whenever resistance is sufficiently lowered from any cause.

Much work has been done in an effort to classify streptococci. By the use of blood agar plates a division into these groups is made:

(1) Hemolytic: those which dissolve the blood corpuscles in the agar, leaving a clear zone around the colony.

(2) Viridans: those which reduce the blood pigment in the culture medium and produce a green color.

(3) Nonhemolyticus: those which have no action on the blood cells in blood agar plates.

The hemolytic streptococcus has the worst reputation although *streptococcus viridans* was a factor in the pneumonias following influenza in 1918. The hemolytic streptococcus causes the disease known as septic sore throat, is found in a very high percentage of cases with diseased tonsils, and is common in otherwise healthy individuals. Observations made at army camps, during the Great War, showed that 36 per cent of measles cases carrying hemolytic streptococci in their throats, developed pneumonia; while only 6.4 per cent of those in whom the streptococcus was not found, developed pneumonia.

Prevalence.—Pneumonia is more prevalent in urban than rural areas, and this is due to the greater opportunity for contact with the organisms which cause the disease. There were 120,000 deaths from all forms of pneumonia in the registration area of the United States in 1920, or more than 10 per cent of all deaths. The appalling toll which pneumonia took from our camps, during the war, was due to

the crowding together of large numbers of susceptibles, in close contact with carriers of the various germs which can cause pneumonia. It is conceded that the bulk of the susceptibles came from the rural districts, and the majority of the carriers probably from the cities. Pneumonia is prevalent at all ages. The acute lobar selects the strong, robust individuals from adolescence to old age; while bronchopneumonia carries off the young babies and children, and is the common form which terminates the lives of so many old people.

Source of Infection.—Source of infection in pneumonia is the secretions from the mouth and nose of cases or carriers of pneumonia. Stillman demonstrated pneumococci in the dust of the sick room, and this fact must be considered. For all practical purposes, the only source is man. The germs may reach the patient from another individual or he may be carrying them, himself, and contract the disease when his resistance is lowered by some other disease or factor.

Mechanism of Infection.—The lungs and bronchial tubes are free from germs in health. The expired air from the lungs is sterile. A small number of germs passing through the trachea would be destroyed, but a larger number of more virulent germs are able to live and multiply below the trachea and produce pneumonia.

The nasal secretions are bactericidal. They destroy many organisms; and under normal conditions are the first line of defense. The tonsils seem to be convicted of affording harborage to germs which cause pneumonia, until the opportunity for the germ to infect the lung presents itself. It is more than likely that the determining factor in infection of the bronchi and lungs is the status of the resist-

ing power of the individual. This may be lowered by many causes, most common of which is attacks of other acute diseases, notably: influenza, measles, and whooping cough. Other factors such as fatigue, exposure, and excesses of various kinds, undoubtedly play a part in pneumonia infections.

The modes of transmission include: droplet infection, direct contact, person to person, including the medium of articles recently soiled with nasal secretions or sputum.

Control.—Cases of pneumonia should be treated with the same preventive care as diphtheria; cases should be isolated, the sputum and secretions from the nose received upon gauze or in paper containers and burned. The same care in sterilizing knives, forks, spoons, and dishes, and the prompt immersion in disinfecting solution or boiling of all bed linen are necessary. There should be the same careful disinfection of hands of nurse and physician, and prompt disinfection of any article which has come in contact with or been used by the patient, to prevent transfer from patient to patient or from the sick to the healthy. The discovery of pneumonia germs in dust of the sick room, while probably not a great source of danger, makes necessary careful, mechanical, moist cleaning of the floors and other exposed surfaces upon which secretions may have fallen.

In lobar pneumonia, as the patient may harbor the virulent germs for about as long as four weeks, it would be wise to continue isolation until the sputum fails to show the germs which cause the disease. The general prevention of the spread of pneumonia depends upon the success of measures which aim to prevent the interchange of sputum and nasal secretions. Education in personal hygiene and regard for other persons' health and safety, can produce results by

cutting down the widespread distribution of these secretions. If every person would attempt to care properly for the discharges from his upper air passage, he would prevent much disease. It would cause some inconvenience in coughing and sneezing, and a great deal of hand washing.

While, very properly, much stress has been placed on coughing and sneezing, do not forget that these respiratory infections are frequently transmitted by soiled hands, and, as shown by Lynch and Cumming, by infected eating utensils.

Vaccines have shown very promising results, but are still in the experimental stage. They would find their greatest usefulness in protecting against the overcrowding, which seems to be inseparable from mobilization of troops, and in similar overcrowding in bunk houses or barracks. The low degree of contagiousness of pneumonia is firmly fixed in the public mind, and general vaccination procedures would not be widely employed, even if they had passed beyond the experimental stage.

CHAPTER VIII

INFLUENZA

Definition.—Influenza is an acute, extremely contagious disease, resembling a severe cold with fever, pain in head, eyes, ears, or muscles. It comes on suddenly after a short incubation period (two to four days), and there is marked prostration or bodily weakness, out of all proportion to the other symptoms. It is often complicated with a very fatal pneumonia and inflammation of the ear or brain. Influenza in a mild form, commonly called La Grippe or "grip," is constantly present; but our attention is focused on influenza because of the outbreaks in epidemic form, which sweep over the entire country. When the epidemic form of influenza overruns countries and continents, it is said to be pandemic.

Influenza is a disease of cycles, in which we have a low endemic prevalence in ordinary years, coupled with tremendous pandemics, which occur at intervals of twenty or thirty years. Usually preceding a real world-wide pandemic, there are pre-pandemic increases in prevalence, amounting to considerable epidemics which have often passed unnoticed except in retrospect. The significance of these pre-pandemic waves is not clear, although there is a natural tendency to connect them with the great pandemic rise which follows.

The special characteristics of the great pandemics are

rapid spread, wide area of distribution, and definite geographical progression along the most widely used routes of travel and trade. Such characteristics undoubtedly marked many of the historical epidemics, tabulated by Hirsch and others, but are especially noteworthy in the pandemics of 1889-90 and 1918. These striking characteristics are lacking in the epidemics occurring in the inter-pandemic periods.

After a great pandemic such as 1889-90 or 1918, there has been apparently, such a thorough seeding of the population with the microbic cause, that for years after outbreaks whose spread is limited may occur anywhere, and if the distribution is wide, this seems more the development from many foci than an orderly geographic progression from a single source. These post-pandemic outbreaks in succeeding years become more local and sporadic, and bear progressively less resemblance to the mode, rapidity, and scope of spread of the real pandemic outbreaks.

History.—While it cannot be proven, there is every reason for believing that influenza is a disease of great antiquity. From a period of four centuries before the Christian era accounts are found of a plague or epidemic, which may have been influenza. The description of these earlier epidemics is too vague to warrant any positive conclusion.

From the sixth to the tenth century, numerous epidemics are recorded, with history of cough and catarrhal symptoms, which are more suggestive of influenza. Hirsch, in his tabulation of influenza epidemics or pandemics, excludes those prior to 1173 as too indefinite and uncertain to serve any useful purpose in his compilation.

Noah Webster describes the first American epidemic, in 1647, and compiles a table of epidemics from 1174 to 1787.

Webster coupled the epidemics in each instance with a volcanic eruption, earthquake, or some unusual climatic condition. Webster made many interesting comments on the epidemiology of influenza. He noted that the epidemics were sometimes limited to the American continent, and that, contrary to usual custom in certain pandemic years, the disease beginning in America spread to the entire world.

As to the date of the first authentic epidemic of influenza, there is much difference of opinion. Hirsch places it as 1173; Webster 1174; Zerviani 1239; Gluge 1323; Schmeich, Haeser, and others, 1387; Thompson, Zulzer, and Seifert 1510. Since 1510, detailed descriptions are more often available, and little doubt exists as to the identity of the first real pandemic in 1580, and the pandemic in the Western Hemisphere 1647, described by Webster. In the eighteenth century, besides rather widespread epidemics in 1709-12, there were decided pandemics in 1729, 1732, 1742, 1757-8, 1761-2, 1767, 1781-2, 1788-90 and 1799.

In the nineteenth century, because of a greater availability of detailed information, we are on firmer ground and we begin to find clear pictures of pandemics, very similar to those known to the present generation. Influenza has tremendous potentiality for disaster on a large scale, and it is certain that future visitations may be expected. As a demonstration of what did happen, and of what may be expected to happen again, we need consider only the appalling disaster of 1918.

Never in the history of influenza, has such a death toll been exacted. It is probable that in the whole history of the world no parallel will be found for the tremendous catastrophe of 1918, if we consider the short space of time and the wide area of distribution, in which the results were

manifest. Statistics can never be accurate in such times of stress, and world-wide estimates are notoriously inaccurate; but the data indicate that in four months, a half million lives were sacrificed in the United States, and that in the entire world, this particular pandemic was responsible for not less than six million deaths.

Prevalence.—With a strong predilection for the winter months, we have influenza with us every year; and in retrospect, we can detect in the mortality statistics, outbreaks reaching epidemic proportions in twenty-two out of the thirty years, from 1889 to 1918.

In 1918-19 the attack rate varied from 15 to 40 per cent, and seemed to be highest in the age group, five to nine; declining in each successive age group, except twenty-five to thirty-four, which exceeded the rate for the group, fifteen to twenty-four. The incidence in 1918-19 was greater in females than males, and the disparity was most noticeable in the ages from twenty-five to forty; indicating, according to Frost, that the females from fifteen to forty-five, were either more susceptible or more intimately exposed to infection than males of corresponding age.

Case fatality in the 1918-19 epidemic was about 2 per cent, and was slightly higher in females under fifteen, and very much higher in females over sixty than in males of corresponding age; but from fifteen to sixty the case fatality was much higher in males.

Cause.—Two years after the epidemic of 1889-90, Pfeiffer, working with purulent bronchitis and bronchopneumonia, observed and cultivated small gram negative bacilli, which he previously had seen in the sputum of cases during the great epidemic. He demonstrated that these organisms could not be cultivated, by the methods used by

other workers in the epidemic of 1889, and this fact was held to explain failure to isolate during 1889. Pfeiffer felt justified in attributing the 1889 epidemic to this cause, which he named the influenza bacillus.

Pfeiffer claimed that he found influenza bacilli in all fresh uncomplicated cases of influenza. He also claimed that they were found only in cases of influenza, acute or convalescent. Since Pfeiffer's time and until the recent pandemic, the great majority of workers have failed to find the influenza bacilli in cases of influenza, in such high percentages as reported by Pfeiffer, and have found *B. influenzæ* in those suffering from measles and other diseases, and in healthy persons.

The experience of most workers in the great pandemic and since September, 1918, has caused them to conclude that the Pfeiffer bacillus is not the cause, and is only a secondary invader. There seems to be a relative unanimity of opinion as to the rôle played by the pneumococci, streptococci, and other organisms commonly found in the upper air passages. Practically all observers consider these as secondary invaders.

Since the great pandemic of 1918, a score of investigators have claimed that the disease is due to a filterable virus. Some of these claims are very plausible, although the demonstrations are based upon infection of laboratory animals only. We cannot say what germ causes influenza. It is due to an undetermined organism which strikes the initial blow, with an attack of influenza. This lowers the patient's resistance, and especially the resistance to respiratory infections, and makes him easy prey for pneumonia.

The pneumonia may be caused by any of the so-called secondary invaders, which lie in wait, the pneumococcus, streptococcus, staphylococcus, Pfeiffer's bacillus, Friedlan-

der's bacillus and others. There are still some observers that regard some of these secondary invaders as the primary cause.

Source and Mode of Infection.—We do not know the germ which causes influenza, whether it is bacterial, protozoal, or a filterable virus; we do know that the source of infection is in the discharges from the mouth and nose of persons, sick with influenza, or carriers of it. It enters the body through the mouth or nose, because the disease is transmitted by direct contact, by the hands, and by coughing, sneezing, or talking (droplet infection).

It is also transmitted by objects freshly soiled with the discharge from the mouth and nose of patients or carriers, especially eating utensils and dishes. Ample evidence is afforded by Vaughan's study of army camps, to show that the infection was introduced by infected individuals coming from an infected to an uninfected camp. Its extreme contagiousness caused many to believe it air-borne. It travels as fast as man and no faster.

Vaughan says:

> Until the possibilities of human transmission have been ruled out, we can place but little reliance on aerial transmission of disease from one community to another. It is not beyond the realm of probability that influenza was transmitted by infected dust raised into the breathing zone within the limited confines of barracks. While such transmission is probable, we are unable to recognize that this was an important factor when compared with the more direct means incidental to intimate human contact.
>
> For the spread of influenza two factors are necessary, first, the presence of the infective agent, bacterial or protozoal or filterable virus or whatever later generations may find it to be, and, second, the presence of susceptible human beings. The transmission of the disease through a community is dependent

upon the rapidity with which the infective material is transmitted from person to person. The more intimate the contact of individuals, susceptible individuals, the more rapid the progress of the disease.

Vaughan (Warren), who made an intensive study of influenza in 1920 in certain selected districts of Boston, arrives at the following conclusions: (1) We find that not only are crowded families more apt to have cases of influenza, but also that crowded families are more apt to have multiple cases. (2) The cleaner the family the less is the likelihood of multiple cases. (3) Families with two or more cases were dirty in a greater proportion than was the standard of all families. Families with no cases and with but one case were dirty in a smaller proportion than the standard.

(4) Single cases predominated in the clean, roomy dwellings, while multiple cases predominated among the dirty and crowded. (5) As a rule there was at least one and usually several individuals in each household who did not contract influenza. (6) As a rule, in the 1920 epidemic, cases of influenza developed in families successively and not explosively. The infection is most frequently brought into the house by the wage earners. Certainly it is always imported from the outside. (7) Sleeping contact is more productive of influenza than are the less intimate forms.

Influenza raced through the army camps. The channels of communication from person to person were multitudinous and unbroken. In the cities the infection was forced to follow a more devious course and its progress was retarded. The total mortality rates in Baltimore and Camp Meade will illustrate this fact clearly.

Week ending	Sept. 20	Sept. 27	Oct. 4	Oct. 11	Oct. 18
Baltimore	14.3	17.0	28.1	69.5	148.3
Camp Meade	6.2	71.8	531.		

Vaughan lays stress on the rôle played by the human hand. He says:

Influenza virus does not pass into the body through the skin of the hand. The hand may become contaminated without danger to the person, so long as the hand does not communicate the infected material to the mouth or nose. Humanity at large makes a common practice of carrying the hand to the mouth and nose many times a day. The human hand enters the mouth on various pretexts—to dislodge food from the teeth, as an unconscious act of nervousness, to carry cigarettes, food, drink, etc. The hand goes to the nose in using the handkerchief.

Lynch and Cumming have shown that of 340 hands of streptococcus hemolyticus carriers, 37 per cent were positive for this organism. Of 40 hands of pneumonia patients, 15 per cent showed pneumococci. Of 95 hands of diphtheria patients, 6 per cent were positive. Of hands of carriers of streptococcus viridans, 100 per cent were positive.

Do these hands transmit germs to inanimate objects commonly handled, such as table tops, door knobs, bedposts, etc.? Of 29 inanimate objects within the range of streptococcus carriers, 24 per cent were positive. Of 14 inanimate objects within the range of pneumonia patients, 21 per cent were positive.

To Lynch and Cumming of the U. S. Army, we are also indebted for very valuable researches emphasizing the part played by infected dishes in spreading influenza. These authors found after a study of influenza incidence in camps that those organizations using mess kits which were washed by the individual in common wash water of lukewarm temperature had much higher morbidity rates than among the units using tableware or mess kits which were boiled. Thus the case incidence of former group was 51.1 per thousand and among the latter 252. The ratio is one to five.

Immunity.—Vaughan's observations on the army camps show that those who had influenza, in the mild epidemic in April, 1918, were immune in September, 1918. Frost's studies in Maryland and American cities show that an attack confers immunity, but that it is fleeting in character, and probably lasts less than a year. The lack of a lasting im-

munity is in striking contrast to measles, which resembles influenza in its extreme contagiousness. Measles attacks children because nearly all others have immunity, conferred by an attack in childhood. Influenza strikes all ages because a previous attack only protects for a few months.

Control.—The first principle of prevention is to break the channels of communication by which the interchange of mouth and nasal secretions is possible; the difficulty of doing this is obvious. Education in personal hygiene will give better control of the cough and sneeze. Greater care can be taught in hand washing and the dangerous habits of carrying the fingers to the mouth. Quarantines are worse than useless; and though isolation is necessary and useful, it does not prevent the spread of influenza. No measures at present known can stop it. It burns itself out, and stops when the susceptible material has been used up.

Some of the measures aimed at overcrowding and close contact, while they do not stop the epidemic, do much good by delaying the spread. This has value in giving time for better organization to insure better medical and nursing care.

Masks were advocated by many, but the weight of evidence shows that wearing of masks had no effect in the spread of the epidemic. The cities which did not close theaters and churches fared quite as well as those which closed them. There seems to be little excuse for closing schools when contact in so many other places *must* go on.

Advice should be given to ensure proper rest, regular meals, and exercise. If taken sick, the patient should be put to bed with the first appearance of symptoms. He should be isolated, not only to protect others, but to protect him from carriers of any of the germs which cause pneu-

monia. All the precautions taken in pneumonia or diphtheria should be observed in the influenza sick room. The sputum and nasal discharges should be received on gauze or in paper containers and burned immediately. Careful disinfection of all articles in contact with or soiled by the patient should be carried out. The doctor and nurse should carefully disinfect their hands before leaving the room. Thermometers and instruments, tongue depressors, etc., should be left in the room and disinfected. Disinfection of dishes and eating utensils should receive special care, and remnants of food should be destroyed or disinfected in the room.

Vaccines.—An immense amount of work has been done in an effort to demonstrate the value of special vaccines; and the literature teems with reports of the results of their use. Most remarkable claims are made by the advocates of the various vaccines. Some of these claims are impressive, until subjected to careful analysis when the lack of controls, or other serious defect, minimizes their value. McCoy has pointed out that, in many of the striking examples, vaccination was practiced after the epidemic had been running for several weeks; and that the vaccinated group was made up of persons, who probably would not have sickened with or without vaccination.

The great majority of the men best qualified to judge the value of vaccines, believe that the vaccines so far recommended and used have little effect, if any, upon either morbidity or mortality. Nevertheless, if in influenza we have a disease in which the initial damage, by an unknown organism, is followed by an attack of fatal pneumonia produced by secondary invaders, then it is logical and reasonable to

attempt to develop protective substances in the body, against these known secondary invaders.

Theoretically, their use is indicated, and a large number of experiments have been made since 1918, with better controls than was possible during the pandemic. They are still in the experimental stage, but there is no reason why they should not be given wide use, if we are unfortunate enough to have another big epidemic.

CHAPTER IX

DIPHTHERIA

Definition.—Diphtheria is an acute contagious disease, with lesions localized in the throat and mucous membrane of the nose or larynx. It is caused by the Klebs-Löffler bacillus, which, growing in the throat, nose, or larynx, causes an intense inflammation with a characteristic grayish exudate or false membrane. The intense local inflammation, and the general symptoms of the disease, are due to the toxin or poison secreted by the bacilli. This soluble toxin or poison has special power for damage to muscle and nerve tissue; and if sufficient is absorbed, the heart muscle is affected, and paralysis of various nerves may follow an attack.

History.—We have fairly accurate descriptions of diphtheria by early Greek writers, and the disease was known through the centuries to the present. It appeared in America soon after the first settlements. A very remarkable study of diphtheria outbreaks was made by Bretonneau in France. He declared croup was laryngeal diphtheria, and differentiated the disease from the sore throat of scarlet fever. His studies placed diphtheria diagnosis on a sound basis; and, although they were made one hundred years ago, they represented the sum of our knowledge until the middle of the nineteenth century, when organisms were noted in the false membrane from the throats of fatal cases of diphtheria.

In 1883, Klebs described the bacillus, which he found con-

sistently in the false membranes of fatal diphtheria cases; and the following year, Löffler isolated the bacillus in pure culture, and studied its effect on animals. Löffler was puzzled by finding his bacillus in the throat of a healthy child and by failing to isolate it from some cases of diphtheria. We now know that the healthy carriers of this germ are relatively common; he may have failed to recover it in all cases, because of the crude technique of the time, or because some of his cases were not diphtheria.

In 1889-90, Roux and Yersin were able to produce in animals the toxic symptoms of diphtheria, using the toxin only, or more precisely, the fluid part of a culture from which all germs had been filtered. The development of antitoxin by Behring and Roux followed, and within five years (1895) was being used by the medical profession.

Prevalence.—Diphtheria is always present in our large cities; but it is most active in fall and winter. Infants under six months are rarely attacked, and 85 per cent of all cases are children under ten.

The Effect of Antitoxin.—Antitoxin, since 1895, has reduced the fatality rate from more than 50 per cent to about 10 per cent. That means that out of every 100 cases, before antitoxin, 50 died; now, with the use of antitoxin, only 10 die. The death rate per 100,000 population for diphtheria has been greatly reduced.

This 10 per cent fatality rate which still persists should be eliminated; and it is due in nearly all cases, to failure to administer antitoxin, or failure to give it early enough. To say that we have reduced the deaths in each 100 cases, from 50 to 10, is not enough, when we have a remedy which should reduce the mortality to zero.

While antitoxin has reduced the mortality from diph-

theria, it has had very little effect on morbidity. In other words, while the number of deaths per 100 cases is fewer, the total number of cases is probably as high as it was ten years ago.

Seriousness of the Disease.—In spite of the reduction effected by antitoxin, 13,000 persons died of diphtheria in the registration area of the United States, in 1920; of this number over 11,000 were children under ten.

With antitoxin available, it is pertinent to ask, why have we any deaths from diphtheria? Carey, of the Massachusetts State Department of Health, reported the results of an investigation of 1,000 diphtheria deaths to determine this point. He found that 31.5 per cent of the children had been sick one week before a doctor was called, and that 11.8 per cent were dying at the time of the doctor's first visit. These figures give one of the principal reasons why we still have deaths from diphtheria, in spite of the fact that we have a remedy (antitoxin) which, given early in proper dosage, will ensure recovery.

Incubation and Period of Communicability.—The incubation period is usually from two to five days; and the patient or carrier may transmit the disease to others, as long as virulent bacilli can be demonstrated in cultures from his throat and nose.

The source of infection is the discharges from the nose and throat of persons sick with diphtheria or healthy carriers, who harbor the germs in their nose or throat. The germs enter the body through the mouth or nose.

Carriers.—The importance of the carrier in diphtheria in spreading the disease can scarcely be overemphasized, although we now differentiate between the virulent and avirulent strains. Even if we exclude those carrying aviru-

lent strains, there still remain a large number of carriers of virulent diphtheria bacilli, in our urban population. The virulent are differentiated from avirulent strains by testing them on guinea pigs. Diphtheria bacilli may persist in the throat or nose of a convalescent or carrier, for varying periods. In half the cases of diphtheria, the diphtheria germs disappear within three days of the disappearance of the false membrane. In about 5 per cent of the cases, the bacilli are still found after two weeks, and occasionally the carrier is still infective after two months.

Modes of Transmission.—The bacilli may be passed directly by contact from the sick to the healthy, as by kissing. The germs may be sprayed into the air, short distances from the patient ("droplet" infection), by speaking, coughing, or sneezing. The bacilli may reach the mouth by means of the fingers or by indirect contact in many ways. Articles soiled by the discharges from mouth or nose of a diphtheria case or carrier, handkerchiefs, toys, pencils, or any of the things which children handle and are constantly placing in their mouths, may be responsible. The multiplicity of ways in which infection from nose, throat, or mouth of one individual may reach the mouth of another, is beautifully shown, by Chapin, in his terse, vigorous language:

> Probably the chief vehicle for the conveyance of nasal and oral secretion from one to another is the fingers. If one takes the trouble to watch for a short time his neighbors, or even himself, unless he has been particularly trained in such matters he will be surprised to note the number of times that the fingers go to the mouth and the nose. Not only is the saliva made use of for a great variety of purposes, and numberless articles are for one reason or another placed in the mouth, but for no reason whatever, and all unconsciously, the fingers are with great frequency raised

to the lips or the nose. Who can doubt that if the salivary glands secreted indigo the fingers would continually be stained a deep blue, and who can doubt that if these nasal and oral secretions contain the germs of disease these germs will be almost as constantly found upon the fingers? All successful commerce is reciprocal, and in this universal trade in human saliva the fingers not only bring foreign secretions to the mouth of their owner, but there exchanging them for his own, distribute the latter to everything that the hand touches. This happens not once but scores and hundreds of times during the day's round of the individual. The cook spreads his saliva on the muffins and rolls, the waitress infects the glasses and spoons, the moistened fingers of the peddler arrange his fruit, the thumb of the milkman is in his measure, the reader moistens the pages of his book, the conductor his transfer tickets, the "lady" the fingers of her glove.

Everyone is busily engaged in this distribution of saliva, so that the end of each day finds this secretion freely distributed on the doors, window sills, furniture, and playthings in the home, the straps of trolley cars, the rails and counters and desks of shops and public buildings, and indeed upon everything that the hands of men touch. What avails it if the pathogens do die quickly? A fresh supply is furnished each day.

Milk Epidemics.—The dicharges from mouth or nose of a carrier, or person with diphtheria, may infect milk if such a person handles milk. The bacilli grow freely in milk; and many epidemics have been reported, in which the source was a carrier or case of diphtheria at the dairy farm.

Immunity.—The immunity following an attack lasts for months or years; second and third attacks have been reported, but Park states that these when investigated are usually found to be some other throat infection. The immunity following an attack is an antitoxic immunity, due to antitoxin developed in the blood of the patient. There seems

to be a natural immunity which develops with age. Infants, up to six months, are immune because of antitoxin derived from the mother before birth. From three to ten years is the most susceptible age period; after ten years susceptibility progressively decreases with the years. This antitoxic immunity permits healthy individuals to harbor diphtheria bacilli in the throat or nose, without having any symptoms.

Active Immunization.—A child is passively immunized by injecting antitoxin, produced in the blood serum of a horse, which has been rendered immune by injections of diphtheria toxin. Active immunization is produced by the injection of small doses of diphtheria toxin, partially neutralized by antitoxin, into the child direct; the child's blood reacts to the toxin and produces its own antitoxin. Each of these methods of immunization has its uses; for prompt immediate immunization of diphtheria cases and those in contact with them, the injection of antitoxin gives immediate protection, which lasts for only a few weeks. The immunity developed in the child itself, by toxin-antitoxin mixtures, takes weeks or months to develop and is, therefore, of no immediate value. This active immunity (by stimulating antitoxin production by the child's own blood) lasts for years.

The Schick Test.—Only those persons contract diphtheria who have no antitoxin in their blood or have it in very minute quantity. In 1913, Schick described a test which would show whether the person tested was immune or susceptible. The test consists of the injection between the layers of the skin, of a very minute quantity of diphtheria toxin. If the person is immune, that is, possesses antitoxin in the blood, this neutralizes the toxin and there is no reaction. If the person possesses very little or no anti-

toxin, and therefore is susceptible to diphtheria, the irritant action of the toxin injected, produces a marked reaction: redness and an infiltration of the skin over an area from 1 to 2 centimeters in diameter.

The value of the Schick test is obvious, especially in connection with active immunization of children. By the Schick test, we can separate the immune from the susceptible children; and the susceptible can then be immunized, by the administration of toxin-antitoxin mixtures.

Control.—The problem in diphtheria is the proper isolation and care of all cases; and this problem is made difficult by the very large number of mild cases and carriers. Obviously, control of all mild cases and carriers is not feasible; but the search should be unremitting, as every case brought under control means prevention of other cases.

I will not hazard a guess as to how many years will pass before parents will isolate and report to the health department, cases not ill enough to call a doctor; yet we will never make real progress in control of these diseases, until parents assume such an attitude.

It is assumed that all cities and towns, and rural districts also, will sooner or later have a health department properly organized. For diphtheria control the success of the department will be in proportion to the support and co-operation given by the householders. There should be laboratory facilities for examining cultures and making diagnosis. There should be public health nurses for checking the control of isolated cases. There should be hospital facilities for cases which cannot be cared for at home, without endangering others. There should be free distribution of antitoxin. There should be an adequate inspection of school children, and schools should not be closed; yet with all these facilities,

unless all cases are reported early, the mild ones as well as those seen by doctors, there will be no real control of the spread.

Examination of Cultures.—About 1892 the discovery of the Klebs-Löffler bacillus, and a special medium upon which the diphtheria bacillus outgrows other germs found in mouth or nose, raised high hopes that all diphtheria cases could be found and isolated, and the disease stamped out. These hopes were not realized. The cultures showed us the extreme frequency of healthy carriers, but these and the mild cases were usually uncontrolled. Cultures showed that from 1 to 2 per cent of children were carriers, and this fact explains why half the cases develop the disease without having had contact with a recognized case. Taking cultures of the entire population is not feasible, and the control of the great number of carriers would be found equally impracticable

Cultures are used to-day for detecting carriers from among those who have been in contact with known cases, or suspected of contact, or of being carriers. It is our main reliance in diagnosis, and in determining when cases may be discharged. Children who are not well, with "colds," "running" nose, husky voice, or sore throat, should be isolated by the parents, without waiting for cultures or doctor; but the parent, if not calling a doctor, should notify the health department and have a culture taken.

Antitoxin.—The hopes raised by the discovery of antitoxin have been realized in the reduction of the case fatality rate. Ten per cent now die where fifty per cent formerly was the rule. The prompt administration of a sufficient quantity of antitoxin will reduce the danger of death to nothing. Antitoxin has not markedly decreased the number of diphtheria cases, and its influence in controlling the spread

of the disease has been less than expected. The prompt use of minimizing doses to contacts prevents many cases, even if it does not greatly affect the general prevalence of the disease.

The first thing to do with a case of diphtheria is to secure a doctor, to give at least 10,000 units of antitoxin to the case; and an immunizing dose of 500 to 1000 units to each of those in contact. Schick tests may be made to determine which contacts are susceptible; but young children in contact should be immunized without waiting for the Schick test.

Toxin-Antitoxin Immunity.—The brilliant work of Park has given us a remedy which makes possible the ultimate elimination of diphtheria, provided the parents of this country will take advantage of the opportunity. Park and his assistants in the New York City Department of Health Laboratories, have perfected the technique of toxin-antitoxin injections, and made demonstrations of what may be accomplished by this method of rendering children actively immune to diphtheria.

The real control of diphtheria depends upon a wider use of toxin-antitoxin immunization, especially in the pre-school-age group. More than 80 per cent of deaths from diphtheria occur in children under five. Not only is the greatest mortality in this group, but the susceptibility to diphtheria is so high, that the use of a Schick test is not always necessary.

Park says:

> Our general opinion is that because of the valuable information obtained it is advisable, when conditions are favorable, but not necessary to give the Schick test to all children after the age of three. At this time they have lost all antitoxin transferred to them by their mothers. The high percentage of positive Schick reactions among children under six years of age and the high

death rate from diphtheria in this age group shows how important it is to protect as promptly as possible all young children against diphtheria. It is a fact that many physicians do not give the toxin-antitoxin injections because they hesitate to use the Schick test. Others give the test improperly or read it inaccurately. Under these and similar conditions it is better to give the injections without the test. Another excellent reason for not delaying for a Schick test before giving the toxin-antitoxin injections to all young children is that only a very mild local and constitutional reaction is produced as a result of the injections.

After the injections of toxin-antitoxin, however, a child should not be pronounced immune to diphtheria until it gives a negative Schick reaction. This test can be made at any time four months or more after the injections. An original Schick test has this great value that, when negative in a child over three years of age, we can assure the parents that the child is probably immune for life.

It is so difficult to reach this age group, one to five, that Park was forced to use the school children in making his convincing demonstration. He had 90,000 school children injected, and had for control a group of 90,000, whose parents had refused the test. These children are still under observation. In the control group the incidence of diphtheria was four times that of the group which received the toxin-antitoxin injections.

In the following table Park summarizes his results:

CASES REPORTED BY PHYSICIANS AS CLINICAL DIPHTHERIA

In Brooklyn:	
26,000 originally Schick-negative children (observation from Oct. 1 to Feb. 15)	2
15,000 Shick-positive children, 3, 2 or 1 toxin-antitoxin injections	4
40,000 control children of same ages	27

In Manhattan:

31,000 Schick-negative children (observation from Oct. 1 to Feb. 15)	3
19,000 Schick positive children, 3, 2 or 1 injections	5
50,000 control children	29

Summary:

57,000 Schick-negative children (observation from Oct. 1st to Feb. 15)	5
33,000 Schick positive children injected with toxin-antitoxin	9
Among a total of 90,000 Schick negative or injected children	14
Among a total of 90,000 control children untreated	56

The cases that did occur in the injected group were mild and doubtful. In the control group some of the cases were very severe.

The precautions necessary in diphtheria in regard to the care of the patient and the sick room, to prevent the spread of the disease, are equally necessary in the other diseases in which the injecting virus is spread by the discharges from the mouth and nose. For this reason, the following standard procedure, by Schereschewsky and Dyer of the Public Health Service, is given in detail:

The Care of Diphtheria Patients

Home care.—Proper care of the diphtheria patient is important in the control and prevention of diphtheria. It is here that the private citizen, if he does his full duty, becomes an efficient unit in the campaign against preventable disease. The communicability of diphtheria and the fact that "carriers" of the germ result from contact with persons sick of the disease, render imperative the strict isolation of diphtheria patients.

The sick room.—The first rule in the care of diphtheria in the home is to place the patient in a separate room. This room should, if practicable, be on the floor of the house the least in use, though its adaptability as a sick room should be taken into account. All

unnecessary furniture should be removed. What furniture is left should be of a kind which may be readily cleansed.

There is no need for fancied attempts at purifying the air by means of hanging sheets wet with disinfectants and the like. If possible, the mattress should be completely covered with a rubber sheet which can be washed from time to time with a disinfectant solution.

Separate linen, bedclothing, etc.—Separate towels, bed clothing, nightgowns, eating utensils, and drinking vessels should be provided for the patient's exclusive use. These should always be kept free from contact with those used by the rest of the family. After being used by the patient, they are to be placed in one of the disinfectant solutions given below or boiled in water.

Attendant for the patient.—The patient should be provided with an attendant who remains with the patient and holds no communication with the other members of the family. This attendant should be the only person caring for the patient or coming in contact with him apart from the attending physician.

Use of disinfectants.—A tub of good disinfectant solution should be at hand for soaking articles which have been used by the patient. A basin of disinfectant should also be provided for cleansing the hands of the attendant. Proper disinfectant solutions are:

(*a*) Two per cent solution of phenol (carbolic acid).

(*b*) Two per cent solution of liquor cresolis compositus U. S. P. (compound solution of cresol).

A 2 per cent solution is made by adding 3 ounces (6 tablespoonfuls) of the disinfectant to 1 gallon of water.

All surfaces soiled by discharges from diphtheria patients should be mopped or flooded with the disinfectant solution.

All articles used by the patient should be soaked for two or more hours in one of the disinfectant solutions or thoroughly boiled. Discharges from the nose and throat of the patient are to be received into pieces of cotton gauze, or old, clean squares of cloth, which are then placed immediately after use into the solution of disinfectant or burned. Partially eaten food is also disposed of by burning.

Care of the attendant's hands.—It is important to remember that

the hands are extremely likely to become infected with diphtheria germs when caring for diphtheria patients and that these germs may then be carried to the mouth. Unnecessary handling of the patient should therefore be avoided. Whenever handling is necessary, the hands should be immediately cleansed in disinfectant solution and then washed with soap and water. This precaution must always be taken by the attendant before eating.

Other precautions for the attendant.—A loose gown or a wrapper should be provided to protect the attendant's clothing. This covering should always be regarded as infected and not sent out of the room until it has been soaked in disinfectant. In the case of female attendants, the hair should be completely covered by a cloth or hood when engaged in caring for the patient. The patient may cough violently in the attendant's face, thus spraying the attendant with the mouth and throat discharges and, possibly, bits of membrane. If this happens the face should be washed at once in disinfectant solution, including the hair if it has been left uncovered. If the hair has been covered, the covering should be placed in the disinfectant solution.

Gowns and head coverings should also be provided for the attending physician. These are kept outside of the room and are soaked in the disinfectant after being used.

Gowns, headdresses, and the like may be thoroughly boiled in water or soapsuds instead of being soaked in a disinfectant solution.

Cleansing the room.—The room should be thoroughly aired two to three times a day. In cold weather the patient should be protected from draft at such times. No sweeping should be done, but the floor and furniture should be wiped with cloths dampened in disinfectant solution. After use the cloths should be soaked in disinfectant or boiled.

Bath after recovery.—After recovery the patient's entire body, including the hair, should be bathed. The patient should then be removed from the sick room and dressed in clean clothes which have not been in the room during the sickness.

Subsequent treatment of the room.—The subsequent cleansing and disinfection of the room after the patient's recovery will, in

cities, be covered by the regulations of the local board of health. When the householder must follow his own initiative in this matter, the following measures should be carried out:

The room should be thrown open freely to air and sunshine. All bed linen, towels, nightgowns, and the like are to be disinfected either by soaking in a disinfectant or by boiling in water. Books and toys used by the patient should be burned. The floors, woodwork, and furniture should be wiped with cloths soaked in disinfectant. Mattresses are best disinfected with steam; otherwise they should be burned. If, however, they have been thoroughly protected by a rubber sheet, after removal of the latter they may be sunned on both sides for a number of successive days.

Duration of isolation in diphtheria.—Persons suffering from diphtheria should be isolated until cultures taken from the throat and nose on at least two successive occasions fail to show the presence of diphtheria germs or until the germs present are shown to be "avirulent" (not able to cause the disease).

Reporting the case.—The efficient control of diphtheria depends upon exact knowledge of its prevalence. It is therefore the public duty of all citizens to report cases of diphtheria to the sanitary authorities and to have the houses in which such cases exist placarded.

It is similarly the duty of the householder scrupulously to observe all regulations made by the local health department with respect to the quarantine of diphtheria cases. All cases of sore throat, especially if occurring in more than one member of a family, should be isolated and steps should be taken to have nose and throat cultures sent to health office for examination.

Protection of food supplies.—When a household in which there is a case of diphtheria is engaged in any occupation having to do with the handling of food, such as the grocery business, dairying, and the like, such occupation should be discontinued until recovery of the patient from diphtheria and virulent diphtheria germs are found to be absent from the recovered case and from the nose and throat of each member of the family. Should the patient be removed to a hospital for contagious diseases, business may be resumed when it is shown that none of the other members of the

family is harboring virulent diphtheria germs, and the necessary cleansing and disinfecting of the premises have been done.

Hospitalization.—From the foregoing discussion of the care necessary for the proper treatment of diphtheria in the home and the precautions required to prevent the spread to other members of the same family, it is quite evident that not all homes are equipped to give this care or render possible the exercise of the proper precautions. Could all cases of diphtheria be promptly hospitalized upon occurrence, we could expect a definite decrease in the percentage of diphtheria cases which end fatally. Furthermore, investigations of diphtheria have definitely shown that the removal of cases of diphtheria to isolation hospitals has a marked effect in reducing the occurrence of other cases in the same family. With the exception of those homes where excellent isolation can be carried out under the care of a trained attendant, hospital treatment is to be recommended. The decision as to hospitalization must be left in the hands of the local sanitary authorities.

CHAPTER X

SCARLET FEVER

Definition.—Scarlet fever or scarlatina is an acute febrile, highly infectious disease usually beginning abruptly with headache, vomiting, chills, sore throat, and rapidly increasing fever. Generally by the end of the second day, a bright scarlet rash appears, much more vivid than the rash of measles. This rash begins on the neck and chest, spreading rapidly over the body. Usually the face is not involved, which is in contrast to the rash of measles. The desquamation or peeling which slowly follows the rash in scarlet fever, is characteristic; large pieces of skin covering considerable areas come away at one time.

History.—There is no doubt of the antiquity of scarlet fever, but the earlier descriptions of epidemics probably included diphtheria cases, and frequently confused measles and scarlet fever. In the sixteenth century, Ingrassias of Palermo and Baillou and Coythar, two Frenchmen, described scarlet fever and differentiated it from measles. In the seventeenth and eighteenth centuries, the disease was epidemic in various parts of Spain and Italy; but the prominence of the throat symptoms makes it probable that diphtheria played some part in these outbreaks. Sydenham described the disease in 1665, and his successor Morton also described it, although he believed it to be a severe form of measles. Epidemics in New England were noted and

described as early as 1735. There has been much discussion of the rôle played by diphtheria in these outbreaks; but on the authority of Vaughan, it may be accepted that the prevalent disease was scarlet fever, with occasional outbreaks of diphtheria as well. These were forerunners of a series of epidemics in New England and the Northern United States, which have continued to the present.

Incidence.—For some reason scarlet fever is much more common in cold or temperate climates than in the tropics. It is also much more prevalent and severe in New England and the Great Lakes Basin, than in our Southern States. It occurs at all seasons of the year, but is apt to be more intense in autumn and winter. It is a children's disease, although adults are not exempt; seventy per cent of all scarlet fever deaths are under ten years of age.

Seriousness of the Disease.—Scarlet fever was responsible for over 4000 deaths in the United States registration area in 1920. It is prone to be followed by middle ear disease, nephritis (disease of the kidneys), and suppuration of the glands of the neck.

Cause.—Scarlet fever is caused by an unknown germ, although for decades claims have been made that the causative organism was a streptococcus. There have been also other claims, but none of these is convincing. The microbic cause of scarlet fever, although unidentified, is known to exist in the secretions from the throat and nose; and is not present in the skin scales, which are thrown off after the rash. The microbic cause is also present in the purulent discharges from the ears and from the glands of the neck. The incubation period in scarlet fever is from two to seven days.

Source.—The source of scarlet fever is always another

human case. The period during which a scarlet fever case is infective or "dangerous" is usually said to be four weeks, if abnormal discharges have ceased; but may be longer, if discharges from the tonsils, ears, or nose, or suppurating neck glands persist.

Modes of Transmission.—It was formerly believed that the infection was air-borne, and that the scales contained the virus. It is now certain that neither belief is justified. It has been demonstrated that, with proper precautions to destroy the discharges of the patient and prevent contact infection, patients can be kept in a ward with non-immune cases without spread of the disease.

Not having identified the microbic cause, we cannot say positively at what period of the disease the discharges are most infective or when they may be considered non-infective. We must consider the discharges from the mouth and nose, and any discharges from ears or glands, as infective during at least four weeks, and longer if abnormal discharges persist.

Scarlet fever is spread by contact either direct or indirect. It may be spread by kissing or direct transference by a cough or sneeze at close quarters into the face of a visitor or attendant. The more common mode of transmission is certainly indirect through articles recently soiled by the patient.

The progress of science has taught us that, in preventing epidemic disease, we must concentrate upon persons rather than inanimate things as carriers of infection, and that the virus of most diseases does not live long outside the body. Scarlet fever is somewhat different from other infectious diseases in these two points: inanimate things recently soiled seem to be the commonest mode of transmission from the

sick to the well, and scarlet fever virus may have a longer life outside the body than the virus of measles and many other diseases.

Until we are able to identify the microbic cause of scarlet fever, we cannot disregard or disprove the frequent statements that scarlet fever virus lives a long time on inanimate things, clothing, toys, etc.

Milk is frequently responsible for scarlet fever outbreaks. A human carrier infects the milk at the dairy and the disease is distributed to the customers with the milk.

Immunity and Susceptibility.—Scarlet fever, while highly infectious, is distinctly less so than measles. One attack usually protects for life, although second attacks in the same individual have been reported. There is a striking difference in Southern cities between the negro and white population; the incidence in negroes is only about one-third of that shown by the white population. We have no biologic weapons, vaccine or serum, with which to combat scarlet fever.

Control.—Failure to prevent the spread of scarlet fever is due to failure to control cases early. Prompt isolation of cases, with thorough disinfection of their discharges, gives good results when practicable. Often cases are not recognized or recognized late, after many have been exposed to the infection. Proper disinfection in the sick room is essential in the control of scarlet fever. The doctor, nurse, or attendant, should put on a gown upon entering the sick room. The gown should be taken off before leaving the room, and the hands carefully disinfected. All bedclothing, linen, or other material in contact with or soiled by the patient, should be immersed in boiling water or disinfecting solution before

being taken from the room, and other precautions should be the same as for diphtheria.

Schools should not be closed because of prevalence of scarlet fever. During an epidemic, a daily medical inspection should be made of the school children, and nurses should visit the homes of children absent from school. In this way many cases are found and prompt isolation secured, which would be undiscovered if schools were closed.

Whether the child is isolated at home or in a hospital matters little, provided the isolation is complete, and facilities exist in the home for proper care and disinfection. If scarlet fever cases are cared for in hospitals, they should be isolated from all other cases, including cases of scarlet fever. This may sound unnecessary, but secondary infection causes high mortality in scarlet fever and a virulent streptococcus, for example, may be transferred from one scarlet fever case to another. If necessary to place cases together in wards, cubicles should be made to separate the cases, and the greatest care exercised by doctors and nurses to prevent secondary infection from any source, including other scarlet fever cases.

Quarantine in scarlet fever should not be less than twenty-eight days, and should be longer if abnormal discharges from mouth, throat, ears, or glands persist. This long quarantine is not only necessary to prevent spread of the disease, but is in the best interests of the patient. The long careful supervision with rest tends to prevent the sequel of nephritis (acute inflammation of the kidneys), which so often follows scarlet fever cases which return to active life too soon.

When scarlet fever is reported, all other children in the house should be excluded from school (unless immune from a previous attack) for seven days from last exposure to the case. The other members of the household, provided the

case is properly isolated, can continue their ordinary vocations, except that of handling milk. Scarlet fever is another example of a disease imperfectly controlled, because of failure of parents to isolate children and report their sickness upon the first signs of deviation from normal health.

Parents usually pay little attention unless the child is ill enough to frighten them into calling a doctor. A great many cases of scarlet fever are mild, with little more than a sore throat. A child comes home from school with sore throat and obviously not feeling well. He should be isolated from the younger children, who often get their infection from the older children of school age; but action of this kind, and active support of the health department, rarely if ever occur.

CHAPTER XI

MEASLES

Definition.—Measles is a very highly contagious disease beginning with fever, reddening (congestion), with swelling (œdema), and increased secretion of the eyelids, with catarrhal symptoms resembling a "cold in the head." These symptoms are followed usually on the fourth day by a skin eruption or "rash," which appears first along the border of the hair, spreading rapidly over the face and neck. The desquamation or scaling following the rash in measles, consists of minute particles or flakes, very different from the "peeling" of large areas of skin which follows the rash of scarlet fever. Sydenham's classic description written in the seventeenth century is good to-day. He described the rash as resembling flea bites on a solid dusky red background.

History.—Measles was probably present in very early times, but its description by early writers was such that it was evidently confounded with smallpox. Sydenham, the great English physician of the seventeenth century, accurately described measles and differentiated it from smallpox. Morton, another English authority, considered scarlet fever to be a form of measles. Noah Webster states that in 1772, measles appeared in all parts of America with unusual mortality, and that 800 or 900 children died in Charleston. There were very severe epidemics with high mortality in England and Scotland, in 1808 and 1811.

Susceptibility and Immunity.—Measles is the most highly contagious of all the acute diseases, with the possible exception of smallpox. The practically universal susceptibility to the disease makes it usually a disease of children. It is relatively rare in adults in cities, because nearly everyone has had the disease in childhood and one attack usually renders the subject immune for life. The universal susceptibility to measles and the lifelong immunity conferred by an attack of the dsiease, are particularly well shown when the disease is introduced into an island population, which has been free from the disease for years.

The Faroe Islands had been free of the disease for sixty-five years when a man from Copenhagen, having been exposed to measles in that city, took ship for the Faroe Islands. A few days after his arrival and fourteen days from the date of exposure in Copenhagen, he came down with measles. From this case measles spread over the entire archipelago; practically all under sixty-five years of age, who were exposed, contracted the disease; those who escaped after definite exposure being over sixty-five years, and presumably had had the disease in childhood.

Incubation Period.—An epidemic starting as this epidemic in the Faroe Islands from one known source, afforded excellent opportunity for studying its manner of spread, period of incubation, and other interesting features. The Danish Government fortunately took advantage of the opportunity and sent a commission, including Dr. Panum, a thoroughly scientific and clear-headed observer. He had many instances of definite exposure and in practically every instance, the period from exposure to the outbreak of the rash was fourteen days.

Prevalence.—Measles has succeeded in impressing itself

upon the casual observer as a benign infection among children, inevitable and annoying, but not especially dangerous. It is a wolf in sheep's clothing, whose power to destroy life is enhanced by this popular fallacy. A disease which caused over 9000 deaths in the United States in 1920, 78 per cent of these being children under five years of age, must be taken seriously. If we reckon the many deaths charged to pneumonia but really due to measles, the toll is greatly increased There are also chargeable to measles many cases of tuberculosis, following an attack of what is popularly regarded as a mild disease.

While measles is a disease of children in the cities, it is somewhat different in the country. The age group attacked in rural districts has a higher age limit; that is, more people in the country escape exposure to measles in childhood, to come down later as adolescents or adults.

Measles has a slightly higher death rate in urban than in rural sections. It is probable that the morbidity or sick rate is also higher in the cities. This is borne out by our experience in the great army camps during the World War. During the winter of 1917-18, Vaughan states that Camp Pike with a division made up of country boys, from Alabama, Arkansas, Louisiana, and Mississippi, had 62 times as many cases of measles as Camp Upton on Long Island, with drafted men from New York City.

How Measles Kills.—Measles uncomplicated rarely kills. Death is usually due to a pneumonia following the measles. Statistics from civil life are unreliable, because of incomplete reporting of this disease. We are able to judge better from army statistics as to the effect of pneumonia on the death rate of measles.

Vaughan gives the following figures:

YEAR 1917

	Cases	*Deaths*	*Per cent fatality*
Measles uncomplicated	44,158	20	0.05
Measles complicated by pneumonia	3,415	927	27.1

The Virus of Measles.—The germ that causes measles has not been identified, but it is a germ so small that it passes through a porcelain filter. It is, therefore, called a filterable virus. It is present in the discharges from the mouth and nose and also in the blood in the early stages of the disease. The virus is less resistant than scarlet fever, and is so frail that it cannot live long outside the body.

Modes of Transmission.—The ordinary source of measles infection is the secretions of the mouth, nose, and throat. All the evidence seems to indicate that the germs enter the body through the mouth or nose. It is reasonably certain, from the experiments which have been made, that the scales which are shed from the skin after the rash do not carry the germs and are incapable of causing the disease.

The extreme contagiousness of measles caused early writers to speak of it as "air-borne." To-day we know that the discharges of the mouth and nose of a case of measles are sprayed into the air in sneezing or coughing, or even in ordinary speaking; and the air, therefore, in the vicinity of the case, if breathed by a susceptible person, is the means of transmitting the infection from the sick to the well. This spraying of the secretions into the air immediately surrounding the patient is called "droplet" infection. The virus may be transmitted by direct contact and by objects very recently contaminated with the virus; but the common means of

transmission is probably through the air by "droplet" infection.

Control.—The control of measles presents one of the most difficult problems which confronts a health officer. This is largely due to two facts: It is probably the most highly contagious of all the infectious diseases, and it is most contagious in the first three days before the characteristic rash appears, when it is rarely diagnosed. Chapin, the dean of our municipal health administrators, says in his classical work, *Sources and Modes of Infection:*

> Measles is a disease which in cities it seems to be impossible to check to any appreciable extent by isolation. In Aberdeen this was faithfully tried for twenty years, 1883 to 1902, but no apparent effect was produced on the prevalence of the disease. Similar failures have been noted elsewhere. During the last half of this period in Aberdeen, when there was far more accurate registration and better control than before, the number of cases rose to 24,254, about fifty per cent more than in the first half of the period. A census of the children in certain schools indicated that from 90 to 93 per cent of children over ten years of age had had the disease. Restrictive measures which protected only seven to ten per cent of the population from attack were then wisely abandoned. The very excellent report of the medical officer of health of Aberdeen discusses the subject very fully. In New York measles was first isolated in 1896, but not until 1902 were the regulations very rigorously enforced. The average death rate from 1895 to 1904 was 2.40, and the highest death rate since 1896 was reached in 1906, when it was 2.69. There is no evidence that the measures adopted in New York have had any more influence on the prevalence of the disease than did isolation and disinfection in Aberdeen. It seems in the highest degree probable that the disease prevails because of the unrecognized but infectious prodromal stage. No amount of isolation after the disease is recognized can atone for the harm done before the diagnosis is made.

It is, nevertheless, possible to secure earlier isolation of measles cases by isolating on suspicion before the diagnosis is made. The intelligent mother often realizes that a child is sick long before a diagnosis can be made, and if mothers can be persuaded to isolate upon suspicion, without waiting for the rash and a positive diagnosis, a great improvement in control will be accomplished. A medical school inspector or school nurse or teacher with a little training, can pick out children with fever above 99 degrees, with suffused or reddened eyes and cold in the head, and many of these are beginning measles cases. As a rule, it is the older child who carries the infection from school into the home and infects the younger children.

In picking out cases of measles before the rash appears, some claim that diagnosis can be made from the so-called Koplik's spots. These are found on the mucous membrane of the mouth and are small irregular spots, bright red in color, with a minute bluish white speck in the center. It is possible for an expert to diagnose a case by finding Koplik's spots, but usually when found, the case has been spreading infection for two or three days. There are other simpler signs which can be more easily found by the inexperienced; and as suggested above, no great skill is necessary to pick what may be incipient measles, because of fever and catarrhal symptoms with swelling (œdema) of the eyelids.

While isolation probably has little effect on the final spread of the disease, it may retard the spread and it should be practiced for other good reasons. It prevents further cases due to the case isolated; and permits of better protection of the sick case from secondary infection. Prompt isolation in bed and good nursing will prevent many cases of pneumonia. As stated, measles rarely kills. It paves the way for invasion

by the germs which cause pneumonia, and it is pneumonia that is responsible for most of the measles deaths.

Further, the greatest mortality is from six months to five years of age. The mortality above five years is low, except in extraordinary circumstances, such as prevailed during mobilization and training in the army camps during the Great War. In ordinary circumstances in civil life, if children can be kept from infection until after five years old, the mortality will not be great.

Besides isolation of the patient, the house should be placarded to prevent visiting. The possibility of the disease being carried by a third person is pointed out by Rosenau, who states that physicians may carry the infection to healthy children. Mild and atypical cases are not so common as in scarlet fever, typhoid, and other diseases. It is also probable that there are fewer "carriers" of measles.

Ordinary precautions in the sick room should include wearing of gowns by everyone entering, gown to be doffed upon leaving, after thorough disinfection of the hands. There should be disinfection in the room itself of all articles exposed to or soiled by discharges. After termination of case, there should be thorough cleansing of the room occupied by the patient, with admission of plenty of sun and air. There is no need for chemical disinfection of the room, because of the short life of the virus outside the human body.

CHAPTER XII

WHOOPING COUGH

Definition.—Whooping cough is an acute, highly contagious disease, whose chief symptom is a characteristic cough. The cough is usually a series of violent explosive efforts to remove an irritant in the bronchial tubes, followed by a prolonged inspiration (whoop). The irritant in the bronchi is furnished by the bacilli (Bordet-Gengou), which cause the disease and are found in the sputum, which is a thick white exudate. The bacilli are most abundant in the first few days; usually after two weeks it is difficult to find them. In certain cases the bacilli persist for longer periods. The incubation period is uncertain, but fourteen days is considered by most authorities as sufficient. The disease is most contagious during the early catarrhal symptoms; before the characteristic "whoop" makes diagnosis easy. For this reason, like measles, the disease is uncontrolled in most cases during its most contagious period.

The period during which the case continues to be contagious is generally conceded to be two weeks after the "whoop" develops. Although difficult to demonstrate the bacilli of Bordet-Gengou after two weeks, there are probably some carriers who harbor the bacilli and spread the disease for a much longer period. Dogs and cats are susceptible and may spread the infection, but they can play only a very minor rôle.

Specific Cause.—Bordet and Gengou described the organism, *bacillus pertussis* or the Bordet-Gengou bacillus, which is now generally described as the cause of the disease. Its claim as the cause is based on the facts that it is found in all cases in the early stage in the sputum, and is not found in any other disease. The blood serum from whooping cough patients gives a specific reaction (agglutination), and the specific fixation of complement test with the *bacillus pertussis*. There is still a minority of observers who are inclined to doubt the causative relation of the *bacillus pertussis*. They admit its constant presence in the early stages, but are slow to admit it is the sole cause, clinging to the old idea that there is a nervous element in the causation of whooping cough. Mallory has suggested that the symptoms of the disease are caused largely in a mechanical manner by the growth of the bacillus in the bronchial tubes; and this is a very plausible explanation of the violent expulsive efforts to get rid of the multiplying masses of bacilli on the delicate, extremely sensitive mucous membrane of the upper air passages. These coughing efforts give the characteristic symptoms and "whoop."

Immunity.—All ages are susceptible, but the disease is so highly contagious that nearly everyone has it in childhood. The immunity from an attack is strong and lasting; as a consequence there are few susceptible people to be found above the age of ten. Of deaths in 1920, in the United States registration area, 55 per cent were under one year, and 95 per cent under five. Even very young children have whooping cough, but it is not so easily recognized: the characteristic "whoop" is absent, the child is cyanosed and, after spasms of coughing, lies exhausted in clammy perspiration.

Prevalence.—The seriousness of the disease is not gen-

erally appreciated, and in this it resembles measles. These two diseases cause the deaths of thousands of children under five years of age, some of which could be prevented by preventing exposure to the disease until after that age.

Over 9,000 children died from whooping cough, in the registration area of the United States in 1920. This does not give the complete toll, however, because many deaths are charged as bronchitis and broncho-pneumonia which are really due to whooping cough. Whooping cough is consistently more prevalent among girls than boys. This has been ascribed to anatomical difference in the larynx of the boy and girl. This greater prevalence in females is not found in the other contagious diseases of childhood.

Whooping cough seems to be particularly severe in the colored race. In Southern cities, the rate is about twice the rate among white children. This is another exception among contagious diseases of children.

A third difference may be noted in the seasonal prevalence. The greatest prevalence is in spring and summer, while other contagious diseases of children prefer colder weather.

Source of Infection.—The fact has been mentioned that dogs and cats are susceptible, but there is no evidence that they really are a factor in the spread of the disease. For all practical purposes the source of the disease is in human cases. If we could control the human cases early, whooping cough would disappear. Unfortunately, we have no diagnostic sign until the characteristic "whoop" appears.

Modes of Transmission.—From its source in the discharges of sputum and nasal secretion of whooping cough cases or carriers, the germs may enter the mouth or nose of susceptible persons in several ways. Besides direct contact as by kissing, there is "droplet" infection and transmission

by articles recently soiled by the patient. Whooping cough is not an air-borne disease in the sense that the infection travels through the air. There is a great deal of explosive coughing and sneezing in this disease, and during these spasms of coughing the germs are sprayed into the air, perhaps projected further from the patient than in other diseases. This makes an ideal "droplet" infection possible in the vicinity of the patient. Pencils, toys, spoons, handkerchiefs, common drinking cups and towels, dishes, and any other articles recently soiled by the patient, are often the means of transmitting the infection.

Control.—The control of whooping cough seems at first glance entirely hopeless. No satisfactory signs for early diagnosis are available. The most constant and striking early sign is a very remarkable leucocytosis, that is, sudden increase in the number of white blood cells or leucocytes in the blood. These may go as high as 30,000. Even this sign is not always present, and involves a count of the blood cells, which is not often made except in a patient seriously ill.

Agglutination tests are not convincing, and with complement fixation tests are applied too late to be of value in early diagnosis. The "whoop" is the first positive diagnostic sign available, and when this appears, the patient has been infecting others for days. In the early catarrhal stage when the disease is most contagious, there is no diagnostic sign which is practical, easy of application, and positive.

The only chance of preventing infection of others by this early case, depends upon a prudent, intelligent mother, who isolates a child with a common cold and keeps the healthy children away from any child that is coughing. It must always be borne in mind, that very young children do not "whoop." This does not mean that with a late diagnosis

preventive measures should not be taken. Even if the case has already exposed many to infection, there is no excuse for permitting further exposures.

Although we know that with present methods, we cannot suppress whooping cough, we should bend every effort toward preventing the infection of all children, until they are at least five years of age.

When a case of whooping cough is discovered in school, it should be excluded from school, but the school nurse or physician should make sure that the health department is notified, in order that the protection of younger children at home may be secured.

Whooping cough should be promptly reported, and the house placarded. Isolation of the patient is necessary, but it is customary to permit the patient more freedom in the open air. In the house, the patient is isolated from the other inmates, and he should be permitted outdoors only in charge of an attendant. Children do much better with moderate exercise in the open air.

The quarantine period should be about six weeks, although the time limit varies in different cities. Usually the quarantine is not lifted until three weeks after the "whoop" develops. The Chicago method is to quarantine in house or yard, first two weeks with attendant, then three weeks more with freedom of the streets with attendant, avoiding crowds, places of amusement, and public conveyances, and wearing on the sleeve a yellow band bearing the words "whooping cough" in black letters.

Vaccines.—Park says, that the injection of bacilli, killed by heat (vaccines), gives protection to a moderate portion of those treated. The immunity does not develop until ten

days to three weeks. The results are not convincing, although a more successful vaccine may be developed.

In the New York City Clinic, among 600 vaccinated children, 368 were supposed to be subsequently exposed to infection; of this number 37 per cent contracted the disease, and 63 per cent did not.

A very good authority, Davison of Baltimore, recently summed up the evidence as follows: "In summing up the prolific and somewhat contradictory literature on the subject, it may be concluded, that injections of Bordet-Gengou bacillus vaccine may have a slight, though unreliable prophylactic (preventive) effect."

The disinfection of the discharges of the patient or of articles soiled by these discharges, and the same technique in the sick room, should be carried out as for diphtheria.

There is the same difficulty in early diagnosis and the same deadly menace to young children in this disease as in measles. The fatality is in inverse ratio to the age. The only hope at present is in the intelligence and eternal vigilance of mothers, who will isolate, without waiting for doctor or diagnosis, all children with catarrhal symptoms, and keep them away from the younger children.

CHAPTER XIII

CEREBROSPINAL MENINGITIS (CEREBROSPINAL FEVER)

Definition.—Epidemic cerebrospinal meningitis is an acute infectious disease, characterized by fever and inflammation of the meninges, or membranes enveloping the brain and spinal cord. The onset is usually sudden, occasionally gradual, with violent headache, described as boring or crushing, as if the head bones were being crushed in a vise. Headache is commonly the first serious symptom, and this early appearance and its intensity differentiate meningitis from typhus and typhoid fever. In cases of sudden onset, vomiting is an early symptom, and may be preceded by vertigo (dizziness). There is great prostration, earlier and more complete, than in typhus or typhoid. Delirium occurs in many cases, and patients may develop a maniacal strength and be very difficult to keep in bed. A very characteristic symptom is the rigidity of the neck muscles. The head is drawn back, and any effort to bring the chin toward the chest causes intense pain. There is also in many cases muscular rigidity of the limbs. These symptoms of muscular rigidity are not developed so early as the headache, but when found, they are almost sufficient to clinch the diagnosis.

History.—It is probable that meningitis came down to our time through the centuries like plague and other diseases, but it is not recognizable as a definite disease entity in the

earlier medical writings. The first clear description of the disease was made by a Swiss physician, in 1805.

There was an outbreak in Medford, Mass., in 1806, and other localities were affected, including Canada, by 1819. From 1836 to 1850, epidemics were reported in France, Germany, Austria, Italy, Spain, Ireland, Denmark, and the United States. Serious epidemics occurred in New York in 1872, and in Boston in 1874.

There was a renewed prevalence in European countries and the United States, in 1901 to 1905. There have been outbreaks reported in the United States almost every year, up to the present.

Prevalence.—The number of persons attacked by epidemic meningitis in any community is usually small, but the number of persons attacked in New York, in 1905, was at least 80 per thousand. The census reports show, that there are from 5,000 to 8,000 deaths in the United States each year. It is a very fatal disease, and we often have a mortality in epidemics of over 50 per cent. It is notoriously a barrack disease and its seriousness as a disease in army camps, during the World War, was second only to pneumonia.

Vaughan and Palmer state:

> Meningitis has appeared in every camp during the six winter months. The prevalence of this disease at Camp Jackson, however, has stood out above that of all other camps. Next to Jackson stand Beauregard and Funston. The rates are 25.7, 12.8, and 9.8, respectively. Next comes Doniphan with 5.0 and the other camps follow with rates at close intervals, the figures receding gradually from this point.
>
> The three camps with the highest morbidity rate likewise have the highest death rate. Beauregard, however, shows a death

rate even greater than Jackson. Again, it is the Southern troops who have suffered most severely from this disease. Those escaping it are the troops from the northeastern and northern sections of the country.

Meningitis, next to pneumonia, has been the most serious disease that the Medical Corps of the Army has had to meet. It is serious by reason of its high fatality and also because this of all diseases shows the greatest excess over the disease in civilian communities. It will be recalled from the early part of this report that meningitis was estimated to be 45 times as prevalent in the Army as in civilian life, whereas the figure for measles was 19 and for pneumonia 12.

The seasonal prevalence is similar to pneumonia and influenza, greatest in winter and spring. It is a disease of children and young adults, males and females being affected equally.

Specific Cause.—Epidemic cerebrospinal meningitis is caused by the *diplococcus intracellularis* described by Weichselbaum in 1887. It is commonly called the meningococcus. The meningococcus is found both before and after death in the spinal fluid and in the exudate upon the meninges. It is also present in the nasopharyngeal secretions of patients and carriers. The bacteriological diagnosis is usually made from examination of the spinal fluid; and the finding of the meningococcus practically settles the diagnosis as cerebrospinal meningitis, and excludes poliomyelitis, encephalitis, and other forms of meningitis. Other germs—pneumococcus, streptococcus, gonococcus, and others—can cause an occasional case of acute meningitis; but epidemic cerebrospinal fever is caused by the meningococcus only.

Source of Infection.—The source of infection is always man. The germs leave the body with the discharges from the mouth and nose. In no other disease have carriers been

demonstrated in such great numbers in proportion to the number of cases. They are of enormously greater importance than the sick cases as sources in spreading the infection. A carrier or a convalescent is infective and capable of spreading the disease, at least as long as the meningococcus can be demonstrated in his nasopharyngeal secretions.

Mode of Transmission.—The mode of transmission is by contact, including "droplet" infection, and articles freshly soiled by the nasal and oral discharges of a carrier. It is a disease in which overcrowding, as in camps, barracks, and tenements, furnishes the necessary close contact which produces the disease by bringing together many individuals, some susceptible and some carrying the germs. It is probable that freshly soiled eating utensils and infected hands may play a rôle quite as important as "droplet" infection through the air.

Vaughan, in regard to the influence of crowding, states:

> We have already called attention to the fact that French medical officers long ago were convinced that crowding favors development and spread of this disease. Our medical officers in the World War had ample confirmation of this fact. Glover found that the number of carriers among English soldiers increased as the barrack beds were brought closer together; that extending the distance between beds did not immediately decrease the number of carriers. With the beds one foot four inches apart there was a carrier rate of ten per cent. If the beds were crowded to one foot the number of carriers rose to twenty per cent, and with further reduction in the space between beds the carriers went as high as thirty per cent.
>
> From observation in our own camps we came to the conclusion that the transfer of meningococci, pneumococci, and streptococci from person to person does not occur as much during sleeping hours as during waking hours. However, it is well to take the number of beds in a barrack as the best measure of the extent to

which crowding is carried, and for this reason insist on a space between beds of at least two and one half feet, and preferably three feet. The smallest crowd in which cerebrospinal meningitis can be transferred consists of two—one a carrier, the other a recipient. If a successful transfer between these two is made they must be close enough so that the spray thrown out in coughing, sneezing, and loud talking from the donor may reach the recipient. Multiply both donors and recipients in the crowd, and you multiply the numbers of successful transfers. It should be plainly understood that the transfer of the meningococcus from one person to another does not require for its success a bedroom or a barrack. As we have had occasion to say in discussing pneumonia and other respiratory infections, men may be dangerously crowded while out of doors. The advantage in a successful transfer of the micro-organism, however, lies within doors, especially in overheated quarters.

In searching the literature of this disease we have found reports of small epidemics in prisons, orphanages, almshouses, and hospitals, but we have seen no report of epidemics confined to public schools. In most schools the excessive crowding essential to the development of an epidemic of this disease does not exist, at least not often. Certainly no child who has been in contact with the disease should be permitted to attend school unless shown by examination not to be a carrier; but the conditions existing in our public schools are, in our opinion, not favorable to the development and spread of this disease. The greater prevalence of cerebrospinal meningitis among negroes and whites in narrow, overcrowded apartments finds its explanation in the greater ease with which the meningococcus is transferred from one to another under these conditions.

Mechanism of Infection.—The incubation period is from two to ten days, but it must be recognized that this period may be prolonged as carriers later become cases. It has been said that all cases have been carriers, and the reason for this statement is this: The meningococcus enters the

body through the mouth or nose and establishes itself in the nasopharynx. If the infected person is immune, the germs get no further than the nasopharynx, and the person becomes a carrier who discharges the germs in his nasopharyngeal secretions. If the infected person is not immune, he some time later comes down with cerebrospinal fever. The mechanism of infection of the brain and spinal cord from the germs established in the nasopharynx is not clear, and the path of invasion is in dispute.

Various theories have been advanced as to the path by which the meningococcus established in the nasopharynx can reach the brain and spinal cord. The sphenoidal and ethmoidal sinuses and the eustachian tube have been accused, but there is no positive evidence that any of these routes is the common one. Abdominal infection has some supporters. The weight of evidence supports the theory that the meningococcus usually travels from its entrenchment in the nasopharynx to the brain and cord by means of the blood. In the cases with symptoms coming on gradually, the meningococcus has been demonstrated in the blood before the meningeal symptoms developed. An infection of the blood by the meningococcus, not sufficient to produce septicemia but sufficient to cause an affection of the heart valves and joints, indicates how easily tissues like the membranes of the brain and cord, for which the meningococcus has such a preference, may be infected.

There is as yet no entirely satisfactory explanation of why, with an infection of the meningococcus in the nasopharynx, one man comes down with the disease while others are unaffected and are simply carriers. The most plausible explanation is on the ground of immunity, yet the immunity conferred by an attack is by no means lasting. It has been

suggested that fatigue may lower resistance, so that infection becomes possible; and this view has considerable evidence to support it.

Mink, of the Navy, observed at the Great Lakes Training Camp, that meningitis carriers in detention, well-cared for, and without hardship of any kind, did not develop meningitis. These same carriers after three weeks, suddenly exposed to hardship and heavy labor, began to develop the disease. Vaughan believes that the difference between the man who develops meningitis, and the man in whom the germs get no further than the nasopharynx, is one of permeability of the walls of the nasopharynx. He says:

It seems to be established beyond a doubt that in some persons—a minority of those exposed—the meningococcus meets with no opposition in its movements towards that tissue for which it has a predilection—the meninges. One man is susceptible to this disease, while another becomes a carrier only, and the difference, as we see it, between the two lies wholly in the degree of permeability of the walls of the nasopharynx. These differences between individuals may lie in inherited anatomic peculiarities of the region of the nasopharynx. This view is supported by the fact that, while as a rule only one member of a family is successfully invaded by the micro-organism, there are instances in which practically every member of the family develops the disease. In some individuals susceptibility may depend upon the presence or absence of lesions in the nasopharynx at the time the meningococcus reaches this locality, or upon the development of lesions at any time during the occupation of this region by meningococci. The wounds in the nasopharynx may be microscopic or they may be gross. In the literature one finds quite a list of cases in which injuries to the skull have been followed by meningitis. Quite naturally, this list is not a long one, because the injury cannot cause specific meningitis unless the organism is present. Again, the injury must be of such a nature, even when meningococci are

present in the nasopharynx, that a path to the brain and cord is opened up. It has been suggested that the meningococcus is assisted in finding its way through the walls of the nasopharynx by some hitherto unrecognized micro-organism. It may also happen that permeability of the wall of the nasopharynx has been made possible by the activity of some bacterium already present in this region when the meningococcus arrives.

We think that the facts which have been brought out in the study of this disease indicate quite conclusively that the meningococcus does not break its way through the nasopharyngeal wall by its own efforts and that it finds its way through these structures only under exceptional conditions. In a nasopharynx which is in normal condition so long as the organism tarries in this locality, the meningococcus is a harmless guest. When the way to the brain and cord is open on the arrival of the meningococcus or is opened during its stay in this locality it becomes a most deadly agent.

Vaccines.—Cerebrospinal fever is so much less contagious, and spreads so much more slowly than the other epidemic diseases that universal or even wide use of a vaccine could not be expected nor justified. Vaccines made by heating the culture of the meningococcus to 50 degrees C. for one hour, seem to have protective value; but this is difficult to demonstrate in a disease to which such a small part of the population is susceptible. It would be useful in mobilization of large bodies of troops, and might be justified in a civilian population in the face of a great epidemic.

Anti-meningococcus serum is made by injecting horses with at first killed cultures, and later live meningococci. After about six months, the horse is immunized and his serum contains antibodies which destroy meningococci and neutralize their poisons. This serum will retain these properties for years.

Lumbar puncture with an aspirating needle and withdrawal of spinal fluid is necessary for diagnosis; and at the same time gives the opportunity for administering the antimeningococcus serum through the same needle. The serum has value if given early. It has reduced the mortality, when given earlier than the third day, from above 50 per cent to below 20 per cent.

Control.—Early recognition of cases and carriers must be the basis of our attempts at control, but the very great difficulty of effecting this result is at once apparent. The most important source of infection and the greatest spreaders of infection are healthy persons who can be discovered only by a careful, rather difficult, bacteriologic procedure. We know that the majority of carriers are uncontrolled, and that the discovery of the minority is largely accidental.

We know that it is impracticable to make cultures of the entire population, and also that quarantine of all carriers, even if they could be discovered, is impracticable in a civil population. In military units or in institutions it is feasible to make a systematic search for carriers and it is also practicable to isolate them until free of the meningococci. In civil life known contacts of cases can be cultured and isolated.

Various sprays containing antiseptics have been used to eliminate the germs from the carrier. These seem to have been uniformly unsuccessful. Carriers lose their germs just as quickly without sprays if kept in the open air or in rooms with a temperature below 65.

Although the prospect of preventing the spread of meningitis is not hopeful, there are certain procedures which should be enforced. Prompt reporting of cases upon suspicion should be required, and the health department should furnish expert diagnostic advice and treatment to

those unable to secure it. The serum should be furnished free. The case should be isolated and the discharges treated with scrupulous care as in diphtheria. The technique of the sick-room attendants and doctors should be the same as for diphtheria. Placarding the house is useful in keeping visitors away. There should be no quarantine of any members of the household who do not come in contact with the sick patient, and whose nasopharynx shows no meningococci.

More lives can be saved by furnishing facilities for early diagnosis and serum treatment, than by any of our present known methods for preventing the spread of disease.

Education of the public in personal hygiene, so that there would be less sneezing, spitting, and coughing in close proximity to other people would reduce the number of carriers and cases. It is probable also that frequent washing of the hands would reduce some of the interchange of nasal and oral discharges.

CHAPTER XIV

POLIOMYELITIS

Definition.—Acute poliomyelitis is an acute infectious disease, which is sometimes followed by paralysis of various groups of muscles. In earlier times, the cases without paralysis were unrecognized and the common observation of paralysis in children gave it the misleading name of infantile paralysis. It is only in recent years that we recognized the fact, that the majority of cases have no paralysis, and have been able to make a diagnosis in such cases.

The disease is caused by an unidentified organism or germ, which passes through a porcelain filter and is classed as a filterable virus. The virus has a special affinity for the nerve tissue of the spinal cord and base of the brain. The symptoms depend upon the parts of the spinal cord or base of the brain affected. The disease attacks children or young adults in apparently vigorous health. There is fever sometimes preceded by a chill, *malaise,* and sometimes stiffness of the neck. Indefinite pains and muscle tenderness are common. There may be coryza and cough or nausea, vomiting, and purging; but symptoms, in the early stage, are very indefinite and diagnosis is difficult except by exclusion of other diseases. The first characteristic sign is apt to be paralysis, and with the indefinite and varied symptomatology, it is easy to understand that cases without paralysis, especially the mild ones, are usually unrecognized.

History.—Infantile paralysis, as it was called, was described by English and German writers over a century ago. In 1840, Heine described infantile paralysis and rightly attributed it to lesions in the cord. The Swedish physician, Bergenholtz, is credited with first recognizing poliomyelitis as an acute infectious disease in 1881.

Very definite additions were made to our knowledge by Wickman, in a Swedish epidemic in 1905. He showed that it is conveyed from person to person by light cases without paralysis, and carriers who have no symptoms whatever. In 1909 Landsteimer and Popper, and in this country Flexner and Lewis, showed that the virus is present in the spinal cord of a child dead from this disease, and that it was a filterable virus.

In 1911 Swedish observers, Kling, Petterssen, and Wernstedt, demonstrated the virus in the spinal cord, intestines, trachea, mouth, and nose of persons dead from poliomyelitis; they later demonstrated the virus in the mouth and nose of living sick cases. Based on these Swedish experiments, Americans and other observers rapidly developed a large amount of additional information. Frost, of the United States Public Health Service, summarizes our present knowledge of the disease as follows:

Means of Transmission.—The essential facts brought out by the experimental studies of poliomyelitis are the following:

The specific cause of poliomyelitis is a minute microorganism, a so-called virus, capable of cultivation in vitro on suitable media.

The virus derived from human cases is pathogenic for apes, producing in them characteristic effects essentially similar to those produced in man. As is the case with many other pathogenic organisms, considerable variations in virulence are noted.

The only animals other than apes which have been found definitely susceptible to the infection are rabbits. The susceptibility

of these animals to the infection appears highly inconstant, and the effects produced are both clinically and anatomically quite different from those produced in monkeys. Römer has found guinea pigs susceptible to an infectious paralytic disease strikingly similar to poliomyelitis, and Neustaedter has recently made observations suggesting that the guinea pig may at times be susceptible to poliomyelitis.

Sources of the Virus.—In the human body the virus has been found: (*a*) *In the tissues and secretions of persons dead of poliomyelitis,* namely, in the brain, the spinal cord, the mesenteric glands, the tonsils, and in the mucous secretions of the naso-pharynx, the trachea, and the intestines, (*b*) *In the secretions of persons acutely ill with poliomyelitis,* namely, in the naso-pharyngeal secretions and in washings from the rectum. The infectivity of these secretions has been demonstrated not only in persons suffering from the clinically typical paralytic forms of poliomyelitis, but also, though less conclusively, in the secretions of those suffering from mild, clinically indefinite, abortive forms.

(*c*) *In the naso-pharyngeal and intestinal secretions of persons convalescent from acute attacks of poliomyelitis.* Although the total number of recorded examinations of convalescents is as yet small, the results of studies by Kling, Wernstedt, and Petterssen suggest that in a very large proportion of persons recovering from poliomyelitis these secretions remain infective for several weeks or even months.

(*d*) *In the naso-pharyngeal secretions of apparently well persons* who have been more or less intimately associated with other persons suffering from poliomyelitis, chiefly in epidemic foci. No figures are available as yet to form an estimate of the proportion of persons who, upon exposure to infection with poliomyelitis, become "carriers," or of the relative proportions of carriers and clinically recognizable cases of poliomyelitis in an epidemic focus. The technical difficulties in the way of demonstrating the virus, involving the injection of filtrates into monkeys, are such that extensive statistics upon this point can hardly be expected in the near future, unless the technique of the demonstration can be greatly simplified.

Outside of the human body the living virus has been demonstrated in nature only in the dust of rooms occupied by poliomyelitis patients and presumably contaminated with their secretions, and possibly (though the demonstration is not fully convincing) upon articles recently handled by persons suffering from poliomyelitis.

In brief, there is at present experimental proof of the following sources of infection: *The secretions of persons ill with poliomyelitis, those convalescent from the infection, and "passive carriers" —that is, persons apparently well who are harboring the specific virus and discharging it in their secretions.*

Avenues and Vehicles of Infection.—As to the avenues through which the virus may enter the human body to cause infection, inferences may be drawn chiefly from experiments upon lower animals. Monkeys may be experimentally infected by injection of the virus directly into the brain, or the subdural space, into the general circulation, the peritoneal cavity, or even the subcutaneous tissue. They may also be infected by *rubbing the virus* upon the scarified mucous membrane of the nose, and even by rubbing it *upon the uninjured mucous membrane.* Also, by the use of massive doses of the virus and under quite artificial conditions, it has been found possible to produce infection by feeding monkeys through a stomach tube.

Of the various methods of infection experimentally shown to be possible, infection through the nasal mucosa appears to be the most constant under conditions which might be expected to be approximated in nature.

Concerning the natural vehicles of infection, experiments performed under laboratory conditions are necessarily somewhat inconclusive. The infectiousness of the nasopharyngeal and intestinal secretions of infected persons and the susceptibility of monkeys to infection through the nasal mucosa indicate very strongly that the disease may be transmitted in nature by such vehicles as may serve to transmit these secretions from infected persons to the respiratory (or digestive) tracts of others—that is, by more or less direct personal contact. The resistance of the virus to the influence of drying and sunlight suggests the proba-

bility of the infective agent being conveyed in dust and in fomites, a suggestion strengthened by the experimental evidence of the infectivity of dust from the sick room.

Experiments showing the possibility of transmitting the infection from monkey to monkey through the agency of a biting fly, *Stomoxys calcitrans,* and in one instance through the bedbug, have added considerable weight to the hypothesis that poliomyelitis is in nature an insect-borne disease. However, since the transmission of the disease through these insects has proven possible in only isolated instances and under highly artificial conditions, these experiments do not warrant the conclusion that stomoxys or other insects play any important part in the natural dissemination of the disease in man.

On the whole, the experimental evidence, taken alone, while not excluding other means of transmission, points to the conclusion that poliomyelitis is a contagious disease, spread from person to person through interchange of infectious secretions, the sources of infection being the clinically definite and clinically indefinite acute cases of poliomyelitis, convalescents, and passive human carriers.

Prevalence.—Poliomyelitis has a worldwide distribution. It occurs in the northern United States in every month of the year, but has a decided tendency to reach its maximum in late summer or early autumn. It is thus an exception to other "sputum"-borne diseases, which are usually worse in fall, winter, and spring. Its seasonal prevalence more closely resembles that of the intestinal diseases, and insect-borne diseases, due to insects most active in summer and autumn. One of the most striking features of this contagious disease is the small proportion of the population affected in epidemics. The number of persons attacked in an epidemic may be less than one per thousand. This means that for some reason, the susceptible material, or non-immune, are relatively a small part of the total population.

Urban and Rural Prevalence.—The prevalence of the disease seems to be greater in rural and suburban districts than in cities. It must be conceded that most of these observations have been made in epidemics rather than in times of endemic or low prevalence.

Age Incidence.—The most consistent epidemiologic characteristic of poliomyelitis is the age incidence. In a mass of contradictory and irregular manifestations only the seasonal prevalence and age incidence are consistent. The great majority of the cases are children under five, a smaller group from five to ten, a few from ten to twenty, and only rarely over twenty.

Assuming that poliomyelitis is a communicable disease, transmitted from person to person, if the rural prevalence is higher in epidemics than the urban, it follows that there must be relatively more susceptible material in the rural districts. It is reasonable to suppose that some immunizing influence, operating more constantly and more actively in the city, cuts down the amount of susceptible material. What is the nature of this influence? Is it due to an increased resistance coming on with age, or is it due to an active immunity acquired by having had the disease in a light or abortive form? It has been demonstrated that convalescents and healthy carriers harbor the virus in the nasopharyngeal secretions for weeks and months. It has also been shown that in apes the nasopharynx, with its close relation, through the lymphatics, with the central nervous system, is an easy portal of entry, and monkeys inoculated intracerebrally have shown that the nasopharynx is also a point of egress for the germs. We have here all the conditions for a widespread distribution of the virus.

In all recent epidemics light and abortive cases have been

shown to be numerous. It is not unlikely that these light cases, which ordinarily pass unrecognized, without paralysis, or paralysis of the most fleeting character, constitute a larger part of the total morbidity than the frank paralyzed cases. In considering the possibility of an active immunizing influence, we must concede also the possibility of variations in virulence, not alone as among individuals attacked, but variations in virulence of the outbreaks themselves. Variations in virulence exist in practically all micro-organisms, and the virus of poliomyelitis is no exception. The difference noted in individual cases in the severity of the attack may be due to a complex set of conditions, involving not only the infecting agent, but also the susceptibility or self-protective power of the individual. Differences in type of an entire epidemic, however, while their cause may be complex, also undoubtedly occur. The fatality rate of epidemics of other diseases varies greatly, it often being lower in centers where the disease has been active over the longest period. Smallpox varies from a mortality of 15 to 30 per cent, to the mild form, practically without deaths, which in the South is called "Cuban itch." Immunity is conferred by Cuban itch, as well as by a severe case of smallpox. It is not improbable that an active immunizing influence is going on constantly, constituting an endemic prevalence of this disease, with relatively few paralyzed cases.

This hypothesis would best explain the age-incidence of the disease, for with a widespread infection, opportunity for immunity is afforded in the first years of life.

Until we know definitely how poliomyelitis is transmitted, it will not be possible to put in effect any real control of the spread of the disease. We must proceed on general principles, requiring that cases be reported and that the isolation

of cases be carried out with rigid technique in the sick room. The virus having been demonstrated in the discharges from the mouth and nose, and intestinal discharges, these must be handled as in diphtheria and typhoid fever, respectively. We know that these frank cases play a minor rôle in spreading the disease, and that the major rôle is played by the healthy carrier, light case, or convalescent from an unrecognized case. With these facts the health officer recognizes the futility of efforts to find carriers or protect the relatively small percentage of the population which is susceptible.

If we had a simple procedure such as the Schick test in diphtheria, that would indicate susceptibility, we would find that one-tenth of one per cent would need protection. It is hoped that a prophylactic or vaccine may also be developed, so that active immunity may be produced in this susceptible one-tenth of one per cent of the population.

The other great need in controlling this disease is a simple diagnostic test which can be applied for the detection of carriers. The present methods are cumbersome, expensive, and impractical for general application; and until further discoveries concerning the virus, its growth and recognition, carriers must go undiscovered and unmolested.

In the face of our rather hopeless lack of exact knowledge, Rosenau gives the following very excellent advice on prevention:

> Until the modes of transmission of the disease are established, however, we can have no confidence in our prophylactic measures, which most resemble the old "shotgun" prescription.
>
> The following measures are recommended: The patient should be isolated as completely as possible in a clean, bare room, well screened to keep out insects. This is a good practice, despite the

fact that the disease shows no tendency to spread in children's asylums, hospitals, and other institutions, or even in the home. The same statement, however, was made of typhoid fever not many years ago. Visiting should be interdicted and only the necessary attendant should be allowed to come in contact with the patient. All discharges, including sputum, nasal secretions, urine, and feces, should be thoroughly disinfected, and special care should be taken that cups, spoons, remnants of food, etc., which may have become contaminated by the patient, are burned, scalded, or otherwise purified.

Towels, bed linen, and other fabrics should be boiled or dipped into a germicidal solution strong enough to destroy the typhoid bacillus. The nurse and physician should observe the same precautions regarding their hands and clothing as are recommended in attending a case of scarlet fever.

The period during which the isolation should be maintained cannot even be guessed at. Children are usually not permitted to return to school for at least three weeks but, if chronic carriers play the important part now suspected, this time would be far too short in many instances.

Since the virus can be killed experimentally by a one per cent solution of peroxide of hydrogen, antiseptic gargles, sprays, and nose washes of this solution are recommended to be used by the patient, the nurse and physician, and other members of the family. In the presence of an epidemic, street and house dust should be kept down by sprinkling, oiling, and other means employed for the purpose. Dust should be allayed whether there is an epidemic of infantile paralysis or not. During epidemics children should be kept away from public gatherings and prohibited from using drinking cups, and special attention given to the diet to prevent gastro-intestinal disorders, for many a case of infantile paralysis starts with a digestive upset.

CHAPTER XV

TUBERCULOSIS

Definition.—Tuberculosis is a general term applied to all disease processes due to the action of the tubercle bacillus. It takes its name from the formation of "tubercles" which is so characteristic of this infection. The tubercles or "little lumps" consist of an aggregation of small epithelioid cells derived from the tissue cells because of stimulation by the tubercle bacillus. These cells are arranged usually around a giant cell in the center and surrounded by white blood cells. The tubercle bacilli are distributed among the cells. At first small, later the "tubercles" become easily visible and attain the size of millet seeds, from which the name "miliary" tubercle was derived. The tubercles break down, and caseous degeneration or "caseation" takes palce, so called because the contents now resemble cheese. This is really death of the cells of the tubercle, caused by the toxin produced by the bacillus.

The coalescence of many tubercles in degeneration produces abscesses and extensive destruction of tissue. Infection with the tubercle bacillus may affect any tissue of the body; but it is most common in the lungs, lymphatic glands, bones, serous membranes (brain peritoneum and pleura), mucous membranes, intestine, and liver.

History.—Smith and Ruffer demonstrated tuberculosis of the spine in mummies of the early Egyptian civilization,

and Hippocrates gave an excellent description of pulmonary tuberculosis. Many others of the older Greek and Roman writers mention and describe the disease.

Our modern knowledge of tuberculosis really begins in 1865, with Villemin's work, which demonstrated the unity of various forms and its transmissibility to animals. He combated the theory of heredity, and indicated a differentiation between the human and bovine strains because of their different virulence for rabbits. In 1882 Koch isolated the tubercle bacillus in pure culture. Theobald Smith, in 1898, established the cultural morphologic and pathogenic differences between the human and bovine strains.

Prevalence.—Tuberculosis has been called "the captain of the hosts of death," and its record justifies the designation. Tuberculosis formerly caused more than 10 per cent of all deaths; and even now, in 1920, it caused over 100,000 deaths in the United States. From 1912 to 1918 the rate was almost stationary, 142 to 150; but in 1919, it dropped to 125.6, and in 1920 to 114.2.

Its wide prevalence will be appreciated if we remember that autopsy records show that practically everyone is at some time infected with tuberculosis. Other factors, notably the individual's resisting power, determine whether the invasion of the body with the tubercle bacillus results in disease and death or merely a healed lesion. It does not regard age or sex, but is much more severe in the Indian and colored races. It destroys in the most productive age, the young adult, and 30 per cent of all deaths between fifteen and sixty are due to tuberculosis. Unless the present rate is further reduced 9,000,000 of the 100,000,000 present residents of the United States will die of tuberculosis.

The following table gives the death rate in the registration area from 1911 to 1920, inclusive:

TUBERCULOSIS (all forms).

Death rate per 100,000 population.
Registration area of the United States.

Year	*Rate*
1911	159.2
1912	149.7
1913	147.8
1914	147.2
1915	146.3
1916	142.1
1917	147.1
1918	150.0
1919	125.6
1920	114.2

Cause.—Tuberculosis is caused by the tubercle bacillus, of which there are at least four varieties: human, bovine, avian, and piscidian. As their names indicate, avian causes tuberculosis in fowls and birds, and piscidian in fish. We are only interested in the human and bovine strains, as the avian and piscidian are not pathogenic for man.

The human tubercle bacillus is a long, slender, rod-shaped organism. Its length varies from one quarter to one half the diameter of a red blood cell (1.5 to 4. microns). Its diameter is about one fifth of its length. It belongs to the group of "acid fast" bacilli; if stained by heat with anilin dyes (fuchsin), it resists decolorization by dilute acids. Although it does not form spores, it is a fairly resistant organism to heat and disinfectants. Low temperatures, and freezing do not kill it; and a temperature of moist heat of 50 degrees C. does not kill unless it be continued for

hours. Half an hour at 60 degrees C. destroys it; and this is the basis used for pasteurization procedure.

The human bacillus, strongly pathogenic for man, has little pathogenic effect on cattle, rabbits, and other animals. The bovine type is very pathogenic for all animals, less so for man. It is pathogenic for man, but much less than the human bacillus. The bovine bacillus is shorter, plumper, and more easily stained.

Cobbett states that the human, bovine, and avian types are descended from a common ancestor, but have acquired different characteristics and are now stable. He ascribes 94 per cent of fatal cases to the human type and only 6 per cent of fatal cases to the bovine. In non-fatal cases, he ascribes 50 per cent of infections in man to the bovine type.

Adami, on the other hand, believes that much of the fatal tuberculosis of adults is a recrudescence of bovine infection acquired in childhood. He believes that the bovine bacillus infection of childhood becomes transformed into the human bacillus type of later life.

Behring and others believe that adult tuberculosis is due to bovine infection acquired through milk in childhood. This theory is supported by many crude facts. The rarity of pulmonary (human type) tuberculosis, and the extreme frequency of bone and gland (bovine type) infection in children is suggestive. Many believe that in the infection with the bovine type bacillus through milk, the bacilli pass from the intestine to the lymph glands, where they remain quiescent until some factor lowers resistance in later life. It has been positively determined that tuberculosis is not hereditary. Children of tuberculous mothers are born free of the disease.

Sources.—The sources of tuberculosis, as indicated

above, are man and cattle. The infection from man to man, in the vast majority of cases, is due to the transfer of sputum containing the germs from an infected person, in some way direct or indirect to the mouth of another. Infection from the bovine source to man is effected through unpasteurized milk. This usually takes place in childhood.

Modes of Transmission.—The infection (tubercle bacilli) enters the human body through inhalation into the respiratory tract or by ingestion (swallowing) into the alimentary canal. The discharges from an infected person may be transferred directly by kissing or by soiled hands, or indirectly through articles recently soiled by the patient. Dishes, spoons, and cutlery are frequent means of spread. J. G. Cumming has shown that spoons used by tuberculous persons with ordinary household washing, retain some of the tubercle bacilli and give some to the wash water and water used for rinsing. Handkerchiefs, toys, food, and hundreds of articles recently soiled, carry infection to the mouths of others.

Close proximity to a tuberculous patient when coughing, sneezing, or even loud talking ("droplet" infection) may cause infection. The germs may also be inhaled with dust contaminated by sputum or feces. Milk carries the infection, especially the bovine type; and it is usually manifest in the mesenteric and cervical glands. The bacilli can pass readily through the mucous membrane of intestine or pharynx and tonsils. The prevalence of tuberculosis in cows, and the failure to pasteurize all milk, expose millions of people and especially children to the infection.

Immunity and Susceptibility.—With the universal distribution of infection, how does anyone escape? Probably few do escape infection or a lodgment of the invading germs

within the body; but another factor, the patient's susceptibility or immunity, determines whether a disease process giving symptoms results. There is a strong natural immunity to tuberculosis in most adults. This immunity is relative, and may be impaired or destroyed by various influences. Other infectious diseases, childbirth, exposure to cold, hunger, and excesses of various kinds, are often determining factors in making a relatively immune person susceptible.

Tuberculosis is becoming a class disease, with improvement in the sanitation of living, and greater understanding of the value of personal hygiene. The rich and the well-to-do are able to protect and fortify themselves, and to retain or increase their relative immunity to this disease. The poor are victims of their environment, unable to improve their diet, take proper rest, or attain proper sanitary housing; their relative immunity is lowered and they become susceptible.

Attempts to produce artificial immunity have been unsuccessful. Koch's tuberculin does not produce immunity, nor do any of the procedures advocated since. The tendency to relapse shows how frail is the immunity acquired in an arrested case; and experience thus far indicates that the immunity may be maintained or increased by general measures, better than by any specific treatment yet discovered.

Control.—One of the most useful principles in control is early recognition of the incipient case, to prevent it from becoming an "open" case, or, if already discharging tubercle bacilli, to convert it into a "closed" case. All open cases should be instructed how to protect others, and those unwilling or unable to observe thse precautions should be isolated.

There should be a constant concurrent disinfection of the

patient's discharges and all articles soiled by them. The sputum is the greatest menace, and should be promptly destroyed after being received on gauze, paper, or in a sputum cup. Special care should be given to sterilizing (boiling) dishes and eating utensils. The floors of the consumptives' rooms should be mopped with strong disinfectant solution.

Sanitaria.—The great value of sanitaria is twofold: to cure incipient cases and to teach them how to remain cured. The graduates of a sanitarium go back to their communities as teachers and the educational effect is considerable. It is manifest, however, that sanitaria can be provided for only a limited number, and that the great majority of consumptives, in the future as in the past, must be treated in their homes.

Hospitals.—Hospitals are necessary for the advanced and hopeless cases, to give them proper care, and to render them harmless to others.

Dispensaries.—A tuberculosis dispensary should have proper equipment for early diagnosis, consultation, advice by experts, and follow up of cases by visiting nurses, who will give special attention to the children.

School Inspection.—Careful school inspection detects early cases and, more important, shows the children who are below par and need outdoor or special treatment. To combat undernourishment is to increase the barrier against infection.

Popular Education.—We know enough now to prevent much tuberculosis. There are two difficulties in applying this knowledge: one is the difficulty in educating large groups of the poorest class, and the other, the poor man's financial inability to follow out the advice given.

The most hopeful drive in education is upon the school child. When we realize that less than half the children reach the high school, it is apparent that the anti-tuberculosis teaching must be given in the lower grades. The open air schools, the added lunch or milk, graduated special exercises, are all most useful instruments applicable through the school inspection.

Improvement in Living Conditions.—The real success in reducing tuberculosis mortality is inextricably bound up with improving the living conditions of the poor; not only the housing, but the factory and industrial hazards must be reduced. Much has been done in industrial hygiene to reduce the health hazards of people who go from the sanitary factory to the filthy, squalid home surroundings. Until housing conditions are very greatly bettered, great further reduction in mortality is improbable.

Practically all the measures which may be expected to reduce tuberculosis are social or economic in character, and their success depends upon the voluntary co-operation of the individual citizen. There is one direct official act which will prevent much infection, especially in children—that is, pasteurization of the entire milk supply.

CHAPTER XVI

SMALLPOX

Definition.—Smallpox, or variola, is an acute, extremely contagious disease with a characteristic eruption of papules, vesicles, and pustules. The disease usually begins with a chill, and sometimes vomiting, followed by high fever. Headache and backache are very common symptoms. The rash comes out on the third day, usually first appearing on face and exposed portions of the body. The papules come first and later become vesicles, containing a clear fluid. The fluid in the vesicles then changes to pus, forming the "pustules." Before the pustule forms, the center sags and a little depression is apparent on the top of the vesicle. This depressed center of the vesicle is called umbilication, and is characteristic of the smallpox lesion. These skin lesions of smallpox contain the virus of the disease and after healing leave "pits" or "pocks" as permanent scars on the skin.

History.—Smallpox is a disease of great antiquity. Ruffer showed that a disease like smallpox probably existed in Egypt about 1200 B.C. as shown by eruptions on the skin of a mummy. It seems to have had its origin in India, and spread to China, then through Asia to Turkey and finally to America. British writers described the disease in the sixteenth and seventeenth centuries and British records of the disease are complete from 1629 to the present.

Inoculation was practiced as a preventive measure by the

Hindoos more than 300 years before Christ; and probably was resorted to by all other nations, at some time since. The first tests of inoculation were successful in England in 1721. The children of the royal household were inoculated and the procedure became popular for a short time. A large number of fatalities followed the early success, and the inoculation fell into disuse. It was practiced by Boylston in a Boston epidemic in 1721, and by Mowbray and Kilpatrick in Charleston, S. C., in 1738.

Smallpox caused terrible havoc during the eighteenth century, over the entire civilized world, and inoculation had little or no effect on its spread. Vaccination was placed on a scientific basis by the classical work of Jenner. In 1796 Jenner demonstrated that an attack of cowpox protected the subject against virulent smallpox. Cowpox originated from smallpox being transmitted to the cow by some human milker. It became modified in severity by its passage through cows, and in human beings is a very mild disease (vaccinia).

Dr. Benjamin Waterhouse of Harvard confirmed Jenner's work by results in his own family in 1800.

Smallpox exacted a tremendous toll prior to Jenner's epoch-making discovery. It was one of the greatest scourges of history and almost exterminated whole communities. It was in these times a children's disease. It was so highly contagious and gave such lasting immunity, that adults rarely had it in countries where it had been prevalent for years. It is estimated that 60,000,000 of people died of smallpox in the eighteenth century. The Spaniards are blamed for the introduction of smallpox into America, and 3,500,000 persons are said to have died from it in Mexico in a short time. Rosenau cites an epidemic in Boston in 1752, which

then had a population of about 15,000. Excluding about 6,000 persons who were immune by previous attacks, only 174 persons who remained in Boston escaped the disease.

Prevalence.—In regard to the existing prevalence of smallpox, and our amazing failure to utilize vaccination fully and wipe out the disease, Vaughan says:

> It will be interesting to collect such information as we are able to find concerning the prevalence of smallpox during the first twenty years of this century. Vaccination by Jenner's method was fairly coming into operation in 1800, this discovery placing in the hands of man the most potent procedure for the prevention of a widely prevalent and loathsome disease. To what extent has man availed himself of this beneficent discovery? In 1900 the death rate from this disease in the registration area of the United States was 1.9 per 100,000. In 1902 it increased to 6.6 per 100,000. Since 1910 it has never reached 0.5 per 100,000. It is a noticeable fact, applicable to other infectious diseases as well, that the death rate from smallpox is much higher in registration cities located in nonregistration states than it is in cities located in registration states. This is to be expected, because the order of admission of states to the registration area is the best possible index of the attention given by the state authorities to the control of infectious diseases. While smallpox is becoming a negligible factor in the death rate of our country at large, it has in certain localities and at certain times displayed a virulence and produced a mortality comparable with those of prevaccination times. At El Paso, Tex., in 1914, the death rate from this disease was 101 per 100,000, and in every year since 1910 there have been two or more outbreaks in localities, causing alarming death rates.
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> As a rule, localities showing unusual death rates from this disease are those which, for the time being at least, include among their inhabitants relatively large numbers of ignorant people. In eleven minor cities showing a death rate of ten or more per 100,000 from smallpox, from the years 1911 to 1917, there has been a large negro population. The migration of this race in large num-

bers to northern cities, notably Chicago and Detroit, has been followed by startling outbreaks of smallpox. Along the Mexican Border the death rate from this disease is kept relatively high by Mexicans. During 1917 there were 110,073 persons vaccinated on the Texas-Mexican Border and even this energetic precedure did not succeed in preventing the introduction of the disease into this country, as is shown by the fact that in the same year the death rate from smallpox at El Paso was 5.8 per 100,000. The smallpox imported into this country from Mexico has been an especially malignant type of the infection. That vaccination and revaccination properly performed may reduce the mortality from this disease to the zero point is shown by the fact that during the World War in no army was the disease anything more than a negligible factor in the mortality lists. In the annual report of the Surgeon General for the year 1919 smallpox is not mentioned, and we fail to find evidence of a single death from this disease in our army or in any division of it, whether located in the continental area, in the Philippines, in the Sandwich Islands, on the Canal Zone, or on the fighting fields of France.

Cause.—The cause of smallpox is probably a protozoal parasite described by Guarnieri in 1892. The parasite is a roughly spherical body, varying in size from about seven-tenths of a micron in diameter to three microns. The period of incubation is from eight to fourteen days, usually about twelve days.

Source.—The source of smallpox is man; the virus is contained in the skin lesions, but smallpox is contagious before the eruption appears. In this early stage the source must be the body discharges, and especially those from the mouth and nose of smallpox cases.

Modes of Infection.—We do not know positively how smallpox is transmitted, but the virus almost certainly enters the body through mouth and nose. It is one of the most highly contagious of diseases, and this fact caused many to

think that it was air-borne. There is no evidence to support this view; and it is not air-borne for any considerable distance. It is probably at times a "droplet" infection, and is air-borne to that extent. Any objects or articles recently soiled by a smallpox patient can serve to transfer the infection. It is also quite possible for flies or insects to transfer mechanically the virus from the skin lesions of a smallpox patient to other individuals.

Control.—The control of smallpox is summed up in one word—vaccination. It is scarcely necessary to more than mention the other measures of secondary importance. If everyone were vaccinated there would be no smallpox; and the only debatable question is whether vaccination should be compulsory.

Plenty of convincing evidence can be produced, showing that with compulsory vaccination smallpox is eliminated; most health authorities believe it should be compulsory. On the other hand, a minority of health officers, comprising some capable administrators, refuse to become excited over opposition to vaccination, on the ground that those who neglect vaccination and oppose it are the ones who must pay the penalty. This is not entirely true and is a dangerous attitude. If the conscientious objector were alone concerned, it would matter little; but these are often propagandists, who carry on with pernicious activity a campaign of anti-vaccination. Unopposed they would secure the support of thousands of careless or ignorant people and create a large unvaccinated, susceptible group of people.

The Pennsylvania law is strong and effective; it requires the exclusion of the unvaccinated child from schools, public, private, or parochial. Rhode Island and Kentucky have compulsory laws and Arizona, Maryland, New Mexico, and

other states require vaccination of school children. Many states have no compulsory laws, and vaccination is done only during or after epidemics.

Smallpox cases should be isolated, but there is no need for that abomination of the last century, the "pest" house. With ordinary sick-room precautions as outlined for diphtheria, and such as should be taken in any contagious disease, smallpox can be cared for in an ordinary contagious hospital. In this disease we do not have to depend entirely on the technique of disinfection in the sickroom, and all persons, contacts, and attendants should be vaccinated. The patient should be isolated in a room, well ventilated and screened. Rosenau distrusts the efficiency of carbolic acid. He gives the following very sane directions:

Bedding, underwear, towels, and other objects should not leave the sickroom unless they are first boiled, steamed, or immersed in a suitable germicidal solution, such as bichloride of mercury, 1-1000, or formalin, 10 per cent. Carbolic acid should not be trusted.

The patient must be regarded as the source and fountainhead of the infection, and measures should be used at the bedside to prevent the surroundings from becoming contaminated. Cloths, cotton, and other dressings that become soiled with the contents of the vesicles and postules after they break should be burned. The urine and feces may be disinfected with chlorinated lime. The sputum and discharges from abcesses should be collected on cheap cloths and burned. As a rule, smallpox patients are not dismissed from quarantine until desquamation has ceased. This may be favored by the use of warm baths and a generous use of soap, also by anointing the skin with vaseline or a bland oil. Special attention should be given to the hair, which should be well shampooed; to the interdigital spaces, and the finger nails, as well as to all folds of the skin, before the patient is released.

CHAPTER XVII

CHICKENPOX; MUMPS; GERMAN MEASLES; SEPTIC SORE THROAT

Chickenpox

Definition.—Chickenpox or varicella is a benign disease, extremely contagious, characterized by an eruption of vesicles on the skin, very mild symptoms, and little constitutional disturbance. It is of epidemiologic interest chiefly because the skin lesions resemble mild smallpox and confusion of the two is not uncommon. It is rarely if ever fatal, and one attack confers lifelong immunity. The incubation period varies in wide limit and is usually given as from fourteen to twenty-one days.

Cause and Modes of Transmission.—The cause of chickenpox is unknown. The virus is probably contained in the lesions of the skin and mucous membrane. The discharge from the mouth and nose is probably infective and is largely responsible for the spread of the disease. It may be spread by direct contact or by dishes, spoons, toys, and any article recently soiled by the patient's discharges.

The children usually have no prodromal symptoms, the first sign being the eruption. The eruption comes out in successive crops (differentiation from smallpox) so that we may have lesions in all stages, papule, vesicle, or pustule at the same time. The fever is usually slight, though high

temperature occasionally is present. It is a disease of children. Its high contagiousness and universal susceptibility ensure that everyone has the disease early. This makes nearly all immune, so that it is rarely seen in adults. Occasionally, a case is seen between the ages of twelve to twenty-one, and very rarely an adult contracts it.

Diagnosis.—It is important to differentiate from smallpox. The absence of the preliminary prodromal symptoms, chill, fever, backache, and headache, suggest chickenpox, as does the eruption in successive crops. At times a very mild atypical smallpox and a fairly severe chickenpox are difficult to differentiate, even for an expert.

Control.—Its resemblance to smallpox makes it necessary to isolate suspicious cases of chickenpox, until the diagnosis is certain and the patient is non-infective; this means until the crusts are thrown off. The patient should be excluded from school and isolated from non-immune persons. There should be concurrent (immediate) disinfection of all articles and materials soiled by the patient's discharges.

Mumps

Definition.—Mumps is an acute, febrile, extremely contagious disease, characterized by an inflammation of the parotid and other salivary glands, and often followed by painful swelling of the testicles or ovaries.

Cause.—The cause is unknown, though Martha Wollstein's work shows it to be a filterable virus. The incubation period is from four to twenty-five days.

Source and Mode of Spread.—The source is always another human case. The virus is contained in the saliva and discharges from the mouth, and possibly from the nose.

The infectivity of the patient probably lasts until the swollen glands return to normal. It is transmitted chiefly through the mouth secretions, either by direct contact or by articles recently soiled by the patient's discharges.

Immunity.—One attack of mumps usually protects for life against a second attack. With its high contagiousness it follows that nearly everyone, in cities and towns, is immunized in childhood.

Mumps may be a serious disease, due to its painful complications; a double orchitis (swollen testicles) may cause sterility. In military camps, it may be a serious problem. Here what is considered a child disease in urban life may have a surprising prevalence in young adult soldiers. It is important not because of the mortality, which is low, but because of the loss of efficiency and large number of days' absence from duty due to it. There were over 200,000 cases of mumps among our soldiers in the World War. In 1918 alone, there were 166,000 cases, an aggregate loss in sickness of 2,894,074 days. In camp life, its mortality is not negligible, and 151 deaths occurred in the army in 1918 charged to mumps; but due in practically all cases to a pneumonia following mumps.

Control.—It has been advocated by good authorities that no effort should be made to prevent persons under fifteen from contracting mumps. The serious complications, especially orchitis, are common after fifteen. This is a disease which lends itself to spread by dishes and tableware in an ideal manner. The saliva and mouth secretions are infective, and careful disinfection of mess kits in camp tends to prevent spread. Rest and abstention from drill and work of any kind, tend to prevent the orchitis.

German Measles

Definition.—German measles or rubella is a mild, infectious disease with a rash or skin eruption. The mortality is negligible. It is chiefly of interest because it is often confused with very mild cases of measles. It is responsible for some of the claims that children have had measles or scarlet fever twice.

The cause is unknown, but it is a distinct disease, with no relation to measles or scarlet fever.

The source of infection is the discharges from the mouth and nose of those suffering from the disease. The incubation period is from ten to twenty-one days.

Transmission.—It is transmitted by direct contact with, or by articles recently soiled by, the patient.

Control.—The disease is so mild that control is urged because of difficulty in diagnosis. A case of measles might be uncontrolled by being called German measles. It is better to take the same precaution in all children before diagnosis is made; namely, isolation and report to the health department. The technique of control should be the same as for measles.

Septic Sore Throat

Definition.—Septic sore throat is an epidemic disease, characterized by an acute inflammation of the throat (tonsils and pharynx) due to the *streptococcus hemolyticus.* It occurs in explosive outbreaks usually traceable to milk, which has been infected by a carrier of the hemolytic streptococcus. The source of the infection is the discharges from the mouth and nose of persons infected with the hemolytic streptococcus. The mastitis (inflammation of the udder) of cows is due to a bovine streptococcus, and has nothing to

do with this disease. A cow's udder may become infected by a human carrier (milker), and then serves as a source, distributing the germs in the milk.

The incubation period is from one to three days. The germs linger longest in the tonsils, and a case or carrier is infective until germs no longer can be recovered from the tonsils by culture.

The mode of transmission is by direct human contact or indirect contact with articles soiled with infected discharges. The epidemics are commonly due to milk, either from an infected udder, or which has been contaminated with the discharges of a human carrier.

Control.—Recognition of cases and carriers by bacteriologic methods, with isolation of patients, and exclusion of carriers from handling of milk or food. Treatment of patient and sick-room as for diphtheria. Pasteurization of the entire milk supply is necessary to prevent milk-borne epidemics.

PART THREE

DISEASES SPREAD BY THE INTESTINAL DISCHARGES

CHAPTER XVIII

TYPHOID FEVER AND PARATYPHOID FEVER

Typhoid Fever

Definition.—Typhoid fever is an acute infectious disease, characterized by fever, an insidious onset, marked abdominal symptoms due to the lesions in the intestines, a tendency to relapse, great prostration and a slow convalescence. For centuries it was confused with typhus, and early attempts at differentiation from typhus gave it the name typhoid.

The history of typhoid fever in the United States has been characterized as a national disgrace; up to 1915 this criticism was not unjust. Sewage-polluted water supplies were so common, prior to 1910, that rates of above 100 deaths annually per 100,000 population were not uncommon; and a total of 25,000 deaths in a year was accepted with complacency.

A vigorous campaign of education by the Public Health Service, and the state and municipal health departments, showing the relation of sewage-polluted water to the prevalence of typhoid fever, has had a wonderful effect in the last ten years, in greatly reducing the disastrous results of water-borne typhoid. These great reductions are due in greatest measure to the installation of water purification plants in our large cities. There is still too much typhoid fever in the United States, because it can and should be entirely eliminated. It is one disease in which we have all the knowledge

and all the weapons necessary for its complete eradication. The prevalence in the United States is shown by the following table:

DEATH RATE FROM TYPHOID FEVER PER 100,000 POPULATION—REGISTRATION AREA OF THE UNITED STATES

Year	*Rate*
1911	21
1912	16.5
1913	17.9
1914	15.5
1915	12.4
1916	13.3
1917	13.5
1918	12.6
1919	9.2
1920	7.8

ANNUAL DEATH-RATES FROM TYPHOID FEVER PER HUNDRED THOUSAND POPULATION IN SOME LARGE AMERICAN CITIES ON THE GREAT LAKES AND THE MISSOURI, MISSISSIPPI AND OHIO RIVERS

City	1906	1907	1908	1909	1910
Chicago	18.3	17.7	15.3	12.6	13.7
Indianapolis	39.2	29.4	26.4	22.3	28.5
Louisville, Ky.	67.7	67.9	44.2	45.0	31.7
New Orleans	29.6	55.5	33.1	28.4	31.5
Detroit	22.3	28.3	22.3	20.5	23.0
Grand Rapids, Mich.	39.1	32.4	31.8	17.2	28.3
St. Paul	21.1	17.1	12.0	18.9	19.5
Minneapolis	32.9	25.9	17.8	21.0	58.7
Kansas City, Mo.	37.8	39.9	35.0	29.3	54.4
St. Louis	11.9	14.1	13.0	16.2	14.9
Omaha	28.2	23.5	22.1	36.8	86.7

City	1906	1907	1908	1909	1910
Buffalo	23.6	29.2	20.7	23.8	20.4
Cincinnati	71.5	46.4	18.6	13.3	8.8
Cleveland	20.2	18.9	12.6	13.3	17.9
Columbus, Ohio	37.1	38.3	110.5	19.6	18.1
Toledo, Ohio	45.0	36.4	40.1	41.7	37.2
Pittsburgh	141.3	130.8	46.6	24.6	27.8
Memphis, Tenn.	42.4	38.8	37.0	48.8	27.4
Nashville, Tenn.	74.4	82.6	59.5	52.0	48.9
Milwaukee, Wis.	30.5	25.7	17.4	21.4	45.7

ANNUAL DEATH-RATES FROM TYPHOID FEVER PER HUNDRED THOUSAND POPULATION IN TEN EUROPEAN CITIES

	Average for 5 Years, 1901-1905	1906	1907	1908	1909	1910
Stockholm	3	2	2	1	0.5	1.8
Christiania	3	4	2	2	1.7	1.6
Munich	4	2	3	3	1.9	1.4
Edinburgh	8	3	3	2	1.2	1.3
Vienna	4	5	3	4	2.8	3.8
Hamburg	4	4	3	4	3.3	4.1
Berlin	4	4	4	4	4.2	2.9
Dresden	4	7	2	6	4.2	2.2
Copenhagen	8	4	2	7	2.7	3.6
London	8	6	4	5	2.2	3.3

In 1910, the writer was placed in charge of an investigation by the federal government (Public Health Service) of the relation of sewage-polluted interstate water supplies to typhoid fever. In his reports, he made the comparison of European and American cities shown above. The European cities had safe water supplies, while most of the American cities were using polluted or dangerous supplies.

In 1910, the rates in fifty largest American cities compared with thirty-three principal cities in Europe were as follows:

TYPHOID FEVER RATES IN LARGE EUROPEAN AND AMERICAN CITIES

Unit of Comparison	Aggregate Population	Deaths per 100,00 from Typhoid Fever, 1910
Thirty-three principal European cities in Russia, Sweden, Norway, Austria-Hungary, Germany, Denmark, France, Belgium, Holland, England, Scotland and Ireland...	31,500,000	6.5
Fifty American cities of 100,000 inhabitants or over.....	20,250,000	25.0
Excess of deaths from typhoid fever in American cities.....		18.5

So that on an average in every 100,000 population we had, compared with European results, 18.5 deaths and at least 180 cases of typhoid fever which should never have occurred. A conservative estimate for 1910 will place the deaths from typhoid fever above 25,000. When we consider that the smaller cities in America have in general higher rates than the larger, that the rural typhoid is high and in many sections higher than the urban, that in the sections not included in the registration area sanitary conditions are probably worse and typhoid fever rates higher than within the area, we are forced to conclude that a general rate of 25 is probably below the actual deaths from typhoid fever per 100,000 population in the entire United States.

The excess of 18 deaths per 100,000 in the urban population alone shows that we have had in the fifty cities mentioned above at least 3,600 deaths and probably 36,000 cases of typhoid fever which were preventable and should never have occurred. For the whole United States the number of cases for each year preventable by methods within our grasp would probably reach 175,000, and the deaths so avoided would total 16,200.

In commenting on these differences in rates, the writer accentuated the fact that in America we must divide our fight against typhoid into two parts: (1) Water-borne typhoid; (2) Residual typhoid; and that in many American cities we had not completed the first step, elimination of water-borne typhoid. These reports also showed that, if we corrected our water supplies and made them safe, we should have typhoid fever rates comparable to the Northern European cities, that is, of less than 5 deaths annually per 100,000 population.

The table on page 158 shows that this condition is being attained. Improvement in water supply has in many cities eliminated water-borne typhoid and these cities have rates in 1920 below 5.

The census reports for 1920 show that there are still many cities which have failed to make their water supplies safe 365 days in the year. After this first step in typhoid reduction, the elimination of water-borne typhoid by furnishing a safe public water supply, there remains the difficult problem of getting rid of what is called residual typhoid.

Cause.—Typhoid fever is caused by the *bacillus typhosus* or typhoid bacillus. It enters the body with food or drink through the mouth, and invades the system through the intestinal mucous membrane. The germs leave the body in

DEATH RATE PER 100,000 POPULATION

TYPHOID FEVER

City	1920	1915	1911
Yonkers, N. Y.	0.	8.8	9.7
Paterson, N. J.	0.7	4.6	7.1
Rochester, N. Y.	1.0	5.9	10.6
Chicago, Ill.	1.1	5.3	10.9
Scranton, Pa.	1.4	13.4	14.5
Boston, Mass.	1.5	5.4	8.8
Worcester, Mass.	1.7	5.5	6.0
Newark, N. J.	1.9	2.9	10.7
Bridgeport, Conn.	3.1	4.8	3.7
St. Paul, Minn.	2.1	7.5	10.6
Grand Rapids, Mich.	2.2	28.0	26.9
Seattle, Wash.	2.2	2.9	10.5
New York, N. Y.	2.5	6.3	11.
Los Angeles, Cal.	2.5	5.8	11.5
St. Louis, Mo.	2.6	7.	16.
Minneapolis, Minn.	2.6	7.9	11.9
Cambridge, Mass.	2.7	1.9	2.8
Lowell, Mass.	2.7	16.4	7.5
San Francisco, Cal.	2.9	8.8	15.2
Columbus, O.	2.9	13.2	13.8
Cleveland, O.	3.	7.5	13.9
Cincinnati, O.	3.2	7.6	11.4
Philadelphia, Pa.	3.4	6.7	14.5
Portland, Ore.	3.5	6.	19.7
Akron, O.	3.7	17.7	36.1
Providence, R. I.	3.8	8.6	12.4
Jersey City, N. J.	4.	6.	7.
Syracuse, N. Y.	4.	5.8	16.3
Albany, N. Y.	4.4	12.2	18.7
Springfield, Mass.	4.6	9.9	18.1
Baltimore, Md.	4.7	20.2	27.5
Buffalo, N. Y.	4.9	9.8	25.1

the intestinal discharges and frequently in the urine. The incubation period is from one to three weeks, with an aver-

age of ten to fourteen days. A patient, a convalescent, or a carrier of the germs can spread the infection as long as the germs are in his discharges. This may extend to weeks and months, and the long-time carrier, with involvement of the biliary apparatus, may be infective for years.

Source of Infection.—The source of infection is always the intestinal discharges or urine of man. There is every gradation in severity in cases of typhoid fever, from severe typical cases to mild atypical cases (walking typhoid) who are scarcely ill, and those who have no symptoms at all. The healthy carriers are the most dangerous source, as they are unrecognized and uncontrolled.

Typhoid bacilli grow well in the urine, and are sometimes present in such enormous numbers that the urine resembles a broth culture of typhoid germs. In preventing the spread of the disease, this measure in the urine is often overlooked and neglected.

The diagnosis of typhoid fever is now made bacteriologically. Before laboratory methods were developed, the clinical symptoms determined the diagnosis. We know now that a majority of the cases do not have typical symptoms. The laboratory diagnosis takes in all cases, grave, moderate and mild, typical and atypical, and those without symptoms —carriers. The most satisfactory diagnostic procedure is culture from the blood of the subject. Five or ten cc. of blood is drawn with a sterile syringe under aseptic precautions from one of the arm veins at the bend of the elbow. The blood is planted in bile, and a growth is possible in twenty-four hours. The germs are tested by the agglutination test with anti-typhoid serum. The typhoid bacilli are in the blood early in the disease, permitting an early diag-

nosis by blood cultures. Ninety per cent of typhoid cases give successful blood cultures in the first week of the disease.

The isolation of the germs from the feces or urine can also be made by streaking the material on plates of Endo's medium (a 40 per cent alkaline agar containing fuchsin) upon which the typhoid bacillus grows in characteristic colonies, which are tested by the agglutination test. These procedures have supplanted the Widal test, in which the patient's blood was tested for agglutinating properties against a known culture of the typhoid bacillus. The Widal test is not likely to be positive until the third week of the disease, and is positive in persons who have had a previous attack of typhoid or have been recently vaccinated.

Vaccines.—The efficiency of anti-typhoid vaccines has been demonstrated beyond question by our army and the armies of foreign nations. It is a harmless procedure, consisting of injection of typhoid bacilli which have been killed by heating. A very small percentage of the millions vaccinated in military service contracted the disease; and among these few, the disease was very mild. The value of vaccines is very great for nurses, physicians, hospital attendants, the personnel of army and navy, and persons in contact with bacillus carriers. As a general measure, it is scarcely feasible for the entire population; and if proper sanitary conditions are maintained in regard to sewage disposal and water supply, it need be used only as an auxiliary to these primary essentials of typhoid prevention.

Bacillus Carriers.—It is probable that a large number of typhoid patients carry the germs through convalescence or after. It has been estimated that from 2 to 4 per cent carry the germs after convalescence. This is a conservative estimate, because many of those eliminated on one of two

negative examinations may still be carriers. Intermittent shedding of the bacilli is very common in typhoid. The stools of a carrier may show negative results for weeks and then suddenly the germs again appear in the feces. This phenomenon has been attributed to storing of the germs in the gall bladder and sudden expulsion of large numbers, after a period during which the intestinal discharges were free of the typhoid bacilli. Reference was made to "Typhoid Mary" (p. 18), and the literature abounds with outbreaks due to long-time carriers; some of them doing damage for many years.

Routes of Infection.—The spread of typhoid fever is attributable to various media which serve to transmit the typhoid bacilli from the intestinal discharges and urine of one person to the mouth and alimentary canal of another. Of these in cities with high typhoid rates, water is unquestionably the most important. Milk plays a part, and other foods, which give the dirty fingers of the carriers opportunity, are responsible in varying degrees of importance. The rôle of flies may have been exaggerated, but they do carry typhoid fever under certain conditions. After water, however, the big outstanding medium of transmission is "fingers."

The eradication of typhoid fever is theoretically possible by the ideal execution of two measures:

1. Safe disposal of human excreta.
2. Disinfection of the hands of those who handle the food and drink of others.

Typhoid would probably disappear if proper and safe disposal of the excreta of the entire population were effected. However, the second measure suggested, hand disinfection, would eliminate the carrier from the problem and, due to

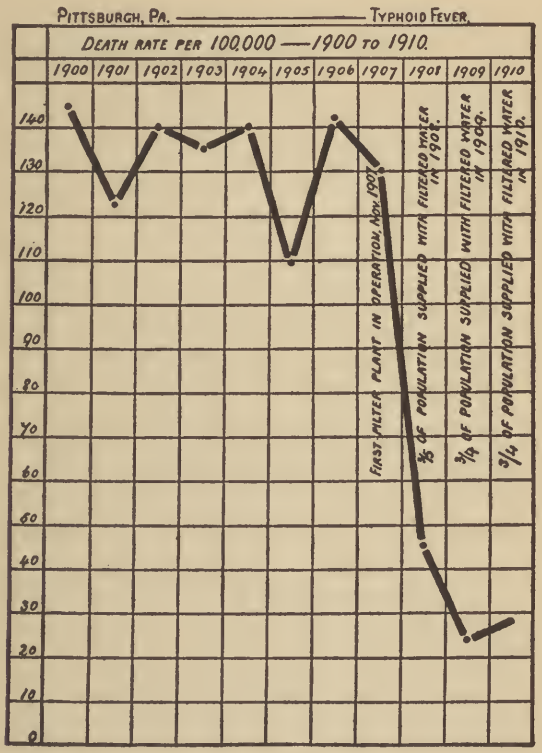
these two measures, typhoid would become a matter of history.

We know practically, however, that proper disposal of excreta of the entire population is an ideal to which we may aspire, but which is still far from attainment. Instead of being able to destroy the infective agent at its source in feces and urine, we are compelled by expediency to attmpt to prevent the entrance of the germs into the human body by making our water, milk, and food supplies safe. We know also that much education in personal hygiene is necessary before we may hope that the individual carrier will protect others voluntarily by disinfecting his hands at the proper time.

Water.—If proof were needed to demonstrate the importance of water in high typhoid rates, hundreds of charts could be made from cities showing the spectacular effect on the typhoid dath rate, of substitution of a pure for a polluted water supply. There is presented here, a graphic illustration of what happened to the typhoid fever death rate in Pittsburgh after the installation of a filter plant for purifying a grossly polluted public water supply.

As an instance of high rate due to failure to furnish filtered water to all the pople, the experience of Pittsburgh is interesting. The filter plant in Pittsburgh was first put in operation November, 1907. But a small portion of filtered water was supplied at first and this was mixed with the unfiltered supply. The amount of water filtered was increased until Octobor, 1908, when the supply of that part of the city between the rivers—about three-fifths of the total population—was filtered. The south side, a little less than one-fifth of the entire population, was supplied with filtered water in March, 1909. The former city of Alle-

gheny, recently annexed, was not supplied with filtered water. This part of the city includes a population of about one-fourth of the entire city. Wards 1 to 20 were supplied with



filtered water. The aggregate population of these twenty wards was 401,622. The typhoid death rate per one hundred thousand in 1910 was 13.4. Wards 21 to 27 comprise the old city of Allegheny and have a total population of

132,283. This section received unfiltered water. The typhoid death rate per one hundred thousand in this section in 1910 was 46.9.

A polluted water supply not only causes typhoid fever directly, but it greatly increases the number of carriers and in this way profoundly affects the number of cases due to contact; the so-called "residual" typhoid. It is a well-known fact that the substitution of a safe for a polluted public water supply does not show its maximum effect at once. The first year there is a marked reduction, and each year thereafter there is a further reduction until in five or ten years, the rate will be below five deaths per 100,000.

There are certain qualifications necessary to make this statement valid. The water purification must be efficient, so that safe water is delivered 365 days in the year, and the water must be furnished to all the people. If purification is imperfect part of the time, or if part of the people use polluted wells instead of the purified public supply, the result will be apparent in the rates. These two reasons, inefficient purification and use of wells, explain why the maximum reduction is not attained more promptly in many cities.

The emphasis placed upon water does not mean that with the installation of a safe water supply all typhoid will cease. The other factors, especially carriers and contact infection, will still constitute a very difficult problem. The existence of these other factors does not absolve municipal officials from their first duty in preventing typhoid, their clear obligation to provide a safe water supply at all times for all the people.

The greatest difficulty was experienced in convincing municipal officials that there was anything wrong with their water supply if the rate for typhoid was below 20 deaths

per 100,000. In may of our Great Lake cities, the health officers were unable to secure a filter plant, but were able to secure the use of hypochlorite or chlorine as a disinfectant. The officials would not admit the need, but the cost of chlorine disinfection was small compared to a filter plant and they consented to its use. The results were startling and convincing. The rates dropped from 20 or more to below 5, and most of these cities are now building proper permanent filtration plants, to take the place of the temporary makeshift, chlorine disinfection.

Milk.—After securing a safe water and proper disposal of human sewage, the next duty of a health department is to eliminate milk-borne typhoid. It is not contended that milk is responsible for more cases than any other factor exclusive of water; but the milk supply can be made safe by official action, and the other factors (contact and carriers) are more difficult to control.

When a milk-borne epidemic does occur, it resembles water-borne typhoid in one particular. It not only directly causes a large group of cases, but it leaves a large number of carriers who increase the rate charged up to contact infections. Milk is infected in most instances by the dirty hands of a typhoid carrier on the farm, or one who handles the milk at some stage between the farm and the consumer. It may also be infected by sewage-polluted water used to wash the cans or to dilute the milk. It is such an excellent culture medium, that germs introduced not only live but increase enormously within the time the milk is in transit.

Milk plays a part in explaining the higher American rates for typhoid fever, because of the difference in amounts consumed and methods of handling. Americans use more milk as a beverage than Europeans, and in Europe the practice

of boiling or heating is almost universal, partly for sanitary reasons, but more often to prevent souring of the milk in the absence of ice. It seems that some of the northern European cities have practically eliminated milk and water and reached the minimum point in residual typhoid, which is just short of complete eradication.

To render milk safe is a more difficult problem in America than in Europe. The Americans use large amounts of milk, and many have a deep-rooted dislike for boiled milk. The heating of milk to the degree necessary to kill pathogenic organisms and its prompt refrigeration seems the only safeguard which can be applied to milk without materially changing its character, which will afford protection to the consumer. There are too many ways in which contamination may reach milk between the cow and the consumer to made milk inspection effective as a safeguard under existing conditions. We hear constantly of the efficiency of inspection, and of preventing further infection by measures taken at the farm, but this is always accomplished after the milk epidemic has occurred. Ideal dairy farms, ideal handling in transit and delivery must and should be striven for, but in the meantime the people should be protected by the means at hand. Inasmuch as we cannot depend as yet upon the individual to protect himself, we must protect him by heating his milk for him. The milk supply should be considered as a public utility and should be controlled by municipal authority as efficiently as the water supply for sanitary reasons. This control necessitates pasteurization or heating for prompt protection of the public. The heating of milk as a prophylactic measure in the campaign against typhoid is comparable to the filtration or treatment of a water supply exposed to pollution.

The Fly.—The fly as a factor in typhoid fever transmission plays a bigger part in the rural districts than in the large well-sewered cities. Measures against the fly by attacking his breeding places should not be deprecated, but with a proper system of sewers and proper treatment of excreta in the sickroom, the fly would be rendered a harmless nuisance so far as typhoid is concerned.

Fingers.—By far the greatest factor in residual typhoid is the more or less direct transference of typhoid fever germs from the fresh feces or urine of one person to the alimentary canal of another. This transference is assisted in certain months by flies, but is consistently effected by the agency of fingers and food during every month in the year.

This contact factor has made possible the persistence of typhoid since prehistoric times, when such things as public water supplies and milk problems did not exist. It depends for its activity upon the close association of individuals with primitive ideals and habits in matters of eating and disposal of excreta.

The savage or primitive man transmitted typhoid by "contact," and the nearer we get to savage or primitive habits of life, the greater the percentage of contact cases, and the greater the difficulty of eradicating residual typhoid.

Fecal contamination of food and drink through fingers and flies is very common and almost constant in certain classes of people in certain rural communities and in sections of large cities. Here the habits of life through ignorance or necessity approach those of the primitive or savage existence, and the refinements of civilization, including the simple act of washing the hands, are considered unnecessary or impossible. Evidence of this is shown by widespread prevalence of intestinal parasites.

Control.—The control of typhoid fever depends upon official measures which ensure a safe water supply for all the people, proper disposal of human excreta, and pasteurization of the milk supply. Besides these official measures, there remains the necessity of controlling all cases of typhoid and carriers of typhoid bacilli, reported and unreported.

The following table summarizes the sources, causes of lack of control, and the remedies for more efficient control:

Possible sources of typhoid.	Causes of lack of control.	Remedy for more efficient control.
WATER,	No filtration or treatment.	Close bacteriologic control; daily quantitative estimation of b. coli; use of chlorine or filtration when necessary.
MILK,	Entire supply is not pasteurized. Large quantities of milk sold in bulk. Many small dealers with dirty shops.	Pasteurization by "held" method in original containers capped and sealed; abolition of trade in bulk milk by small dealers.
UNREPORTED CASES:		
(*a*) Light.	No physician called.	Education of the householder. Wide employment of city physicians.
(*b*) Atypical.	Physician uncertain as to diagnosis.	More prompt use of the municipal laboratory. Disinfection of excreta of all suspicious cases pending diagnosis.
(*c*) Typical.	Physicians called late. Tardy report by physician.	Education of the householder. Better co-operation of practicing physician with the health officer.

Possible sources of typhoid.	Causes of lack of control.	Remedy for more efficient control.
(*d*) Carriers.	Presence undetected.	Greater effort to detect carriers. More extensive use of laboratory. Instruction of carriers in personal hygiene. Carriers must not engage in certain avocations.
Reported Cases:	Lack of adequate instruction.	Visiting nurses.
	Failure or inability to understand instruction.	Hospitalization.
	Failure to carry out instructions.	Hospitalization.
	Failure to continue disinfection of excreta of convalescents still infective.	Disinfection should not be discontinued until laboratory examination negative twice in succession. Instruction of convalescents and carriers. Registration of typhoid convalescents and carriers.

Typhoid fever must be considered in the same category as Asiatic cholera. After we have made our water and milk supplies safe, the fight against typhoid fever, to be successful, must be made with the same vigor and along the same lines as a successful campaign against cholera. There is very little excuse for even such low rates for typhoid as prevail in our large cities. The difficulty of getting rid of the last vestiges of infection when the rate is below 2 deaths per 100,000 is conceded, but with a rate above 10 there is little excuse for failure to wage an active eradication campaign against typhoid as a dangerous contagious disease.

Control of Cases.—Adequate and early control of cases depends upon co-operation among the health officers, the practicing physicians, and the householders.

Duty of Physicians.—The moral obligation of physicians to report promptly cases of typhoid fever is beyond question. The legal obligation is another matter. Personally, I do not believe it is often necessary to appeal to the law to secure prompt reporting of cases. Physicians should bear in mind that no quibble over diagnosis will justify delay in reporting a suspicious case. It is perhaps a great deal to expect that the very busy practitioner will give his valuable time to patient instruction of stupid or illiterate patients. There is only one course of procedure, however, which absolves him from this duty, namely, prompt reporting to the health office, after which the obligation devolves upon the officials to acquire and maintain proper control of the patients' excreta. There has been a great change in the attitude of the profession toward the laboratory as an aid to diagnosis. The very common occurrence of so-called atypical cases is recognized, and there is a tendency to make a much wider use of the clinical laboratory as a routine procedure. The mere fact that doubt exists as to the exact diagnosis does not justify the failure to disinfect excreta. In all suspicious cases the practicing physician has the moral obligation to secure the disinfection of the patients' excreta through his own efforts or to notify the health officials so that they may secure this result pending the definite establishment of the diagnosis. While valuable time is sometimes lost by tardy reporting of cases by physicians, the danger resulting in cities is probably much less than that which follows failure to acquire control of cases

which are never seen by a physician or are seen late in the disease.

Education of the Householder.—For this reason adequate and early control of the typhoid fever excreta necessitates the co-operation of all citizens as well as all practicing physicians with the health officials. Failure to acquire control of typhoid excreta is most common in the poorest families, where through poverty a physician is called only in extremity. This necessitates for its correction the education of the houeholder in the danger of the excreta of the sick. A sincere effort must be made to awaken in him a sene of his responsibility to others. He must be convinced that he has an obligation to prevent human discharges from reaching food and drink. If the poor can be educated to this point, and encouraged to report illness, before it becomes serious, to the city physicians, there is hope for acquiring early control of typhoid cases. Disinfectants should be furnished free and the campaign of education continued in the family by the visiting nurse.

Hospitalization.—The ideal place, from the sanitarian's standpoint, for caring for typhoid fever patients is in the modern hospital. Hospitalization may be unnecessary, and results quite as good may be secured in some homes where intelligent nursing is possible. For the poor, hospitalization is better for both the patient and the community. There are intermediate types of homes where expert nursing is not possible because of the expense, but where the inmates are capable and intelligent, needing only expert instruction in handling excreta. It is in this type of home that the visiting nurse finds her greatest usefulness.

Hospitalization should be insisted upon in two classes of cases:

1. Inability to understand and carry out proper precautions at the bedside.

2. Failure or refusal to carry out these precautions. The physician, health officer, or visiting nurse should make every effort to secure proper handling and disinfection of excreta. If this is not possible for either of the reasons given above, hospitalization should be effected.

Carriers.—There should be a wider use of municipal laboratories, not only in establishing prompt diagnosis, but especially in exercising control of convalescents and carriers. The day has passed when a practitioner in justice to himself and to the public may make diagnoses on the clinical picture alone. Beyond the mere fact of establishing a positive diagnosis, the laboratory is invaluable in controlling the danger from convalescents and carriers.

The carrier is the greatest menace when engaged in the purveying of milk and food. It seems feasible that an examination for carriers might be exacted as a prerequisite to granting a license to furnish food and drink to the public. Persons engaged in occupations involving the handling of food and drink in dairies, restaurants, hotels, lunch rooms, etc., should at least be subjected to a Widal test.

Control of carriers is a very difficult phase of the problem, independent of the difficulty in finding them. The most hopeful solution lies in the education of the individual in personal hygiene. The carrier must be made to understand that he is a menace, and to realize his obligation to protect his fellow citizens by disinfecting his hands, and by exercising care in disposing of his excreta.

There should be a registration system for carriers. They should be allowed full liberty subject to certain provisions. They should not engage in vocations involving the handling

of food or drink, and their whereabouts should be known at all times to the health officials.

The procedure outlined in regard to hospitalization of cases and control of carriers suggests measures which may seem arbitrary and restrictive of personal liberty. As direct compulsory measures, they may not be feasible, but these results may often be attained without compulsion by educational effort. As it is largely a question of personal hygiene or family hygiene, the citizen must be educated to do voluntarily without compulsion things which under compulsion seem to him an infringement upon personal liberty and rights.

Treatment of Patient and Sick Room to Prevent Spread of the Disease.—The room should be screened and stripped of all unnecessary hangings, rugs, or other equipment. Disinfect stools, urine, or sputum, immediately upon its exit from the body. The bedpan should contain a small quantity of strong disinfectant solution, and the feces and urine should be thoroughly mixed with twice its bulk of a compound cresol solution, one part to fifty parts of water, or some equivalent disinfectant. The covered mixture should stand one hour before being emptied.

Everything which comes in contact with the patient or his discharges must be disinfected before it is taken from the room. There should be a large tub containing compound cresol solution for reception of towels, bedding, and other soiled fabrics. Spoons, dishes, glassware, and other articles used by the patient, should be immersed in disinfectant solution or boiled before being taken from the room.

Doctor, nurse, or attendant should put on a gown upon entering the room, and remove it just before leaving. Every person just before leaving and after removal of the gown,

should thoroughly disinfect the hands. The disinfecting basin for hand washing should be placed on a stand or chair just inside the door. The disinfectant solution should be one teaspoonful compound cresol solution to a pint of water, or other good disinfectant of equivalent strength. First soak the hands in the disinfectant solution for two minutes, next scrub with soap, water, and brush, and lastly immerse the hands again in the disinfectant solution for at least one minute. Rubber gloves are a useful safeguard in handling the patient. The gloves and brush should be kept in disinfectant solution.

No typhoid fever patient should be discharged, and the sick-room disinfection precautions should be continued, until repeated examinations of the feces and urine fail to show the typhoid bacillus present. Discharged patients and convalescents should be warned of the possible danger of infecting others, and instructed to wash and disinfect their hands thoroughly after using the toilet or before handling food.

Paratyphoid Fever

Definition.—Paratyphoid fever is an acute, febrile, infectious disease, very similar to mild cases of typhoid; very often it is possible to diagnose one from the other, only by laboratory tests. Paratyphoid has a worldwide distribution; epidemics occur, but not so frequently as in typhoid, and great disasters due to water-borne infection never occur in paratyphoid.

Cause.—Paratyphoid is caused by the paratyphoid bacillus, which resembles the typhoid bacillus very closely. Typhoid germs are not highly fatal to laboratory animals. Paratyphoid is extremely virulent for guinea pigs and mice.

The differentiation is best made with specific serum and the agglutination test.

Source.—The source is the discharges from bowels and bladder of persons infected with the paratyphoid bacillus. Healthy carriers play an important part in spreading the disease.

Modes of Transmission.—The modes of transmission are the same as for typhoid: water, milk, food contaminated with discharges of infected persons, by personal contact, or by indirect contact with articles recently soiled with such discharges.

The incubation period is four to ten days, and a patient should be considered infective until at least two examinations of his stools fail to show paratyphoid organisms.

Control.—The control and prevention measures are identical with those employed against typhoid fever.

CHAPTER XIX

ASIATIC CHOLERA

Definition.—Asiatic cholera is an acute specific epidemic or endemic disease, due to the presence of the *Vibrio Choleræ Asiaticæ* and of its toxic products (Koch, 1883) presenting usually the symptoms of violent purging, vomiting, muscular cramps, suppression of urine, great fall of blood pressure, subnormal temperature, and collapse.

History.—It is considered probable by historical students that Asiatic cholera has existed as an endemic disease in the delta of the Ganges for centuries. From this endemic home the disease became epidemic in neighbouring districts, and we have positive evidence of epidemics of Asiatic cholera in Goa (1543), Pondicherry (1768), Calcutta (1781), and other parts of India. In the nineteenth century the disease first assumed pandemic proportions and spread from India over Asia, Africa, Europe, and America.

In 1817 the disease spread over all of India and during the period from 1817 to 1837 had become a world disease and a world problem.

Since 1817 six distinct pandemics of cholera are distinguishable.

First pandemic	1817-1823
Second pandemic	1826-1837
Third pandemic	1846-1862
Fourth pandemic	1864-1875

Fifth pandemic	1883-1896
Sixth pandemic	1902-1910

The history of these various pandemics is singularly similar. From its endemic home in India by means of the pilgrims and the caravan routes the disease was carried to Afghanistan, Turkestan, Persia, and Arabia. Egypt was usually infected from Arabia. From Egypt, especially after the employment of steamships for sea travel, the infection was rapidly carried to Mediterranean ports of Turkey, Italy, Spain, and France. Pilgrims carried infection from Mecca to Syria, Palestine, Asia Minor, and Russian territory about the Black Sea. Russia also received infection direct from Central Asia over the great caravan routes from Persia, Afghanistan, and Samarcand to the lower Volga and Baku. Infection of Germany and Austria is traceable to Russian and Polish sources. The danger to the United States at present, as in the past, lies in the importation of the infective agent in the person of immigrants from the great European seaports.

Etiology.—In 1883 Robert Koch demonstrated that Asiatic cholera was an intestinal disease caused by a comma-shaped bacillus found in the contents or walls of the intestine. He demonstrated the connection between an infected cistern and a severe outbreak of cholera.

Morphology of the Cholera Vibrio.—In stained preparations the cholera vibrio is a short, slightly curved rod about 1.5 microns in length and 0.4 micron in width. Great variation from type is more apt to be found in old cultures which have been kept on artificial media for long periods. Cultures not more than twenty-four hours old and freshly isolated from stools will be generally found to conform to the type described above.

The motility of the cholera vibrio is remarkable. In a hanging drop they shoot through the field with great rapidity. That the motility is due to a single flagellum has been demonstrated by Kolle and his co-workers; noncholera vibrios have frequently two to six flagella.

The cholera vibrio does not form spores; hence is easily killed.

Typical Severe Case.—If cholera should invade the United States, the first case discovered would probably be a severe or fatal one. It seems wise, therefore, to describe the post-mortem appearance and symptoms of a typical severe case. The appearance of the cadaver in cholera is characteristic. Cyanosis is marked. The skin is dry and the abdomen retracted; the eyes are sunken, half closed, and lusterless. *Rigor mortis* sets in early, and muscular movements, especially of the fingers, may occur for some hours after death. Upon opening the body the tissues are found to be dry and the serous cavities without fluid. The blood exuding from the organs on section is thick and tarry. The right heart and venous system are engorged with blood. The left heart and arterial system are empty. The skin of the fingers and toes is shriveled—the so-called "washerwoman's fingers." The injection of the small intestine gives it a pinkish color which is very striking by comparison with the large bowel or with normal intestines. Upon opening the peritoneum the intestines will be found to be without luster, resembling "ground glass" and covered with a peculiar sticky material which, with the diffuse rosy color of the small intestines, is pathognomonic of cholera.

Typical cholera begins with profuse watery stools. The fecal character of the first stools is soon lost and the discharge assumes the appearance of thin rice water, with

flocculi or granules of mucus suspended therein. The first vomited material may contain food, but later the vomitus is thin and watery, resembling rice water. Muscular cramps in the abdomen and limbs cause great suffering and the spasmodic knotty contraction of muscles is characteristic of the disease. There is a very rapid shrinkage of the soft tissues of the body, due to the enormous loss of fluid, and evidenced by falling in of the cheeks, sunken eyes, shriveled fingers and toes, and general emaciation. There is usually complete suppression of urine and bile. Respirations are rapid and shallow. The body surface is cold and covered with a clammy sweat. The surface temperature falls 4 or 5° below normal, but the rectal temperature may show 38 to 40° C. The pulse becomes rapid, feeble, fluttering, and then imperceptible at the wrist. Cyanosis is marked; the face, and especially the fingers and toe nails, assuming a bluish tint. The voice is reduced to a whisper. These symptoms are sometimes followed by complete collapse and death. This may occur at any time before the expiration of twenty-four hours.

Bacteriological Diagnosis of Cholera.—In combating cholera, our sheet anchor is the exact bacteriologic diagnosis. Diagnosis by means of the agglutination reaction and Pfeiffer's phenomenon permits us to differentiate cholera from toxic gastro-enteritis, ptomaine poisoning, and other diseases resembling cholera. It further enables us to diagnose Asiatic cholera when the classical symptoms are absent or masked, or in those cases in which the patient presents no symptoms whatever (bacillus carriers). In other words, this exact diagnosis obviates the necessity of fighting in the dark, and enables us to concentrate our efforts upon finding and rendering innocuous foci of infection.

The fecal material is planted in peptone broth and from this, after eight hours' growth, agar plates are made which give cholera colonies in eighteen hours more.

These colonies are tested as follows: A dilution of 1 to 200 of an agglutinating cholera serum having an agglutinating limit or titer of not less than 1 to 1000 should be used. A drop of this dilution should be placed at each of three equidistant points upon a clean glass slide. These drops upon the slide are numbered 1, 2, and 3. A portion of colonies correspondingly numbered is transferred from the plate to the drops of diluted serum by means of a straight-pointed platinum wire. The diffuse cloudiness effected in the drops of serum remains permanent in the case of noncholera vibrios, but if the vibrio be cholera the familiar phenomenon of agglutination is microscopically apparent. The diffuse cloudiness gives place within a few minutes to a clear fluid, containing numerous floccules in suspension. The droplets soon dry in the air and may be fixed and stained, when the characteristic vibrios may be seen stained in clumps.

Prevention of Cholera.—Before considering prophylactic measures it is necessary to consider how cholera is spread. The infective agent in cholera is found only in the stools and vomit of persons who have in some way taken cholera organisms into their alimentary tract. The organisms may have been ingested directly into the stomach with food and drink, or at least the germs must have gained entrance to the mouth in some way. Cholera is spread from place to place by individuals, carrying the cholera vibrios in their intestine and more or less sick with cholera. Where the distance between infected points is considerable, the disease is probably carried by man, and by man alone.

Cholera is an absolutely preventable disease, and theoretically a case of cholera properly cared for should not result in further spread of the infection. The spread of cholera is primarily due to one of four factors: (1) Bacillus carriers. (2) Unrecognized light or atypical cases of cholera. (3) Failure to find or report cases early. (4) Carelessness in carrying out precautions, or failure to take such precautions.

The Bacillus Carrier.—The bacillus carrier is an individual carrying cholera vibrios in his intestine and yet who exhibits no signs of the disease. The writer has never known a bacillus carrier to harbor cholera vibrios for longer than twenty days and the great majority lose their vibrios in less than ten days. However, many observers have found them present for longer periods, although all agree that the long-time carrier is the exception and not the rule.

During times of epidemic bacillus carriers are numerous, and the writer found 6 to 7 per cent of carriers among healthy individuals living in the infected neighborhoods in Manila. When cases are few, the so-called sporadic cases, hundreds and even thousands of stools may be examined before the first carrier is found. The fact that the bacillus carrier may harbor the cholera vibrios as long as sixty-nine days illustrates how quarantines may be passed and an apparently inexplicable outbreak be explained. The danger from the bacillus carrier depends upon his habits and the sanitary conditions of the community in which he finds himself. If he deposits his stools in a modern flush closet in a city in which disposal of human excrement is properly effected, and if he washes his hands frequently enough and at the proper time, he is harmless. His urine contains no vibrios. He may find himself, however, in a community

with no proper system of disposal of excreta, or in spite of the existence of such system he may deposit his stool where flies or other insects have access thereto, or deposit it in a place from which a well or other source of water supply becomes infected. He may fail to wash his hands after defecation and with his dirty fingers infect the food or drink of others.

In these ways the bacillus carrier is the greatest menace, and because of presenting no symptoms necessitates for our protection the safe disposal of the feces of the entire population.

Mild or Atypical Cases.—Unrecognized, light, or atypical cases of cholera, or failure to carry out the necessary precautions, or carelessness in carrying out these precautions in recognized cases, are responsible for the spread of cholera by permitting the infective material contained in the stools or vomit to get beyond control. A cholera stool improperly cared for may be deposited where flies and other insects may carry the vibrios to exposed food or drink. In communities without a safe water supply the stool may be deposited in or near a source of water supply. Milk may become contaminated either by flies or by washing the containers in infected water. Kitasato asserts that the vibrios will live only until the milk sours. There is some question about this, but in any event this duration of life would be quite long enough to permit milk to spread the disease.

Vegetables and fruits growing close to the ground are sometimes fertilized by human excrement. They may also be irrigated by infected water, and if eaten raw may thus be a means of spreading cholera.

Vaccines.—The immunity in cholera is relatively short; possibly one to two years' artificial active immunity can be

produced by injections of the cholera bacilli killed by heat. This vaccine seems to protect against attack in the large majority; and in those vaccinated, who are attacked, the mortality is lower. Vaccines are unnecessary against cholera in communities which have proper disposal of sewage, safe water supplies, and proper sanitary conditions. When these conditions do not exist or cannot be secured, as in armies in the field, in camps, and for persons constantly exposed as physicians, nurses, and attendants in cholera hospitals, vaccines have a useful field.

Isolation of the Patient.—A patient with cholera or suspected of having cholera should be isolated immediately. The room or ward should be rendered fly-proof by screening. In the room with the patient there should be a tub or other large vessel containing 5 per cent solution of carbolic-acid crystals for the immediate reception of soiled linen.

The stools and vomit of the patient should be disinfected at once by adding an equal volume of 5 per cent carbolic-acid solution, 5 per cent formaldehyde solution, or milk of lime. The mixture should be covered and allowed to stand for two hours before ultimate disposal. There should also be a washstand and basin just inside the door of the room, and every person before leaving the room should be required to wash and disinfect the hands thoroughly with a one per cent solution of lysol or other good disinfectant.

Gowns should be put on upon entering the sick room and should be taken off just before disinfecting the hands and leaving the room. These gowns when soiled should be placed with other soiled linen in the tub of carbolic-acid solution.

Disinfection.—There should be a thorough surface dis-

infection of *every room in the house* in which a case of cholera or suspected cholera is found.

The infection of cholera is not air-borne and is not likely to be found higher than a man can reach, so that this disinfection is effectively secured by mechanical cleansing of the walls and floor with disinfecting solution (2½ per cent carbolic acid, 1 to 1000 bichloride solution). This disinfection should not only be performed after the death or removal of a patient, but of course should be more or less continuously carried out in the sick room or hospital ward by mopping of the floor and washing or spraying the walls with the disinfectant solutions above described.

The cholera organism is easily killed by drying and by heat, and infected objects may either be immersed in 5 per cent formalin or 5 per cent carbolic-acid solution, or disinfected by dry heat or boiling water.

All remnants of food about a cholera house should be destroyed by burning. Drinking water or other beverages should be disinfected and disposed of. Cutlery, kitchen utensils, crockery, etc., are best disinfected by boiling.

Observation of Contacts and Precautions to be Taken with Them.—After isolation of the patient and disinfection of the premises, the contacts or persons who have been in contact with the sick one must be cared for.

The hands of the contacts and such clothing as may have been exposed to infection must be disinfected, and the contact visited twice daily for a period of five days. During these five days there should be at least two examinations of the stools of each contact, one as soon as possible after discovery of the initial case and the other before discharge from observation. Should either of these examinations prove positive for cholera vibrios, the contact must be iso-

lated at once and the same precautions taken as in any other case of cholera. Until two vibrio-negative reports are received, stools of contacts and their hands are to be disinfected precisely as in actual cholera cases.

Convalescents should have three vibro-negative reports of stools examined on successive days and should never be discharged upon one single vibrio-negative report.

CHAPTER XX

DYSENTERY

Dysentery includes two diseases: one, bacillary dysentery, an acute epidemic disease due to the dysentery bacillus; the other, amebic dysentery, a tropical disease without epidemic manifestations, caused by a single-celled protozoal parasite called the *ameba histolitica.*

Bacillary Dysentery

Definition.—Bacillary dysentery is an acute, febrile infectious disease, characterized by frequent bloody evacuations of the bowels. The stools may exceed fifty per day with painful straining (tenesmus) and prolapse of the rectum.

History.—Dysentery was mentioned in an Egyptian papyrus in 1600 B.C. Hippocrates also described the disease. It is the disease known for centuries as camp, jail, ship, and famine dysentery. In the nineteenth century, it was a common disease of institutions. It was not until 1898 that the cause of bacillary dysentery was isolated by the Japanese scientist Shiga. In 1900, Kruse isolated a similar bacillus from dysentery cases in Germany; and in the same year Flexner, Strong, and Musgrave, in dysentery cases in Manila, found the Shiga bacillus and a closely related bacillus which differed slightly in fermenting a wider range of sugars.

Prevalence.—Bacillary dysentery has a wide distribution in temperate, subtropical, and tropical climates. In war, it formerly caused great havoc, and in our Civil War was one of the major causes of disability. In more recent wars, where greater attention was paid to sanitation, it has played a minor part. It is difficult to estimate its prevalence in this country. A great deal of bacillary dysentery is included in the census reports, under the classification diarrhea and enteritis, under two years. In addition to these, there were over 3500 deaths reported as dysentery in 1920, in the registration area of the United States.

Cause.—Bacillary dysentery is due to the bacillus of dysentery, which resembles the typhoid bacillus in size and shape, but differs in not being motile and in having a weaker action in fermenting sugars. The incubation period is from two to seven days; and the patient is dangerous until the germs can no longer be demonstrated in the stools.

Source and Modes of Transmission.—The source of dysentery is the intestinal discharges of man. It may be transmitted in any of the ways which serve to spread typhoid fever infection. It is very often water-borne. Milk is an ideal medium of transmission; and all the contact factors which give opportunity for dirty fingers to do their dirty work in typhoid, operate to spread dysentery. The disease germs are not found in the urine, and carriers do not carry the germs for long periods as in typhoid; but the carrier is a considerable factor in spreading the disease. The disease is very often spread by infected hands of attendants on the sick, especially where the attendant prepares food for others.

Control.—The frequency of the evacuations makes complete disinfection of dysentery discharges difficult; but the greatest single measure in prevention is thorough hand dis-

infection. If the attendants on the sick thoroughly disinfected their hands before eating, or before handling food of others, prevention would be accomplished. Disinfection of the stools and articles soiled by the patient, and treatment of the sick room, should be the same as for typhoid fever.

Vaccines.—Vaccines have been prepared by using cultures of the bacilli killed by heat, but severe toxic reactions have followed the injection, and vaccination is not generally practiced.

Amebic Dysentery

Definition.—Amebic dysentery is a chronic infectious disease due to an ameba (*entameba histolitica*) which begins insidiously, develops slowly, and is often followed by abcess of the liver. It is a tropical disease and does not occur in epidemic form. The action on amebic dysentery of emetin, an alkaloid derived from ipecac, is comparable to the action of quinine in malaria. The entameba motile form is promptly killed by emetin. The encysted forms are resistant, so that emetin cures the attack by destroying the active motile forms. It does not prevent carriers or relapses, because it does not destroy the resistant encysted forms. These perpetuate the disease, although carriers are sometimes cleared of these forms by irrigations of nitrate of silver and quinine.

Prevention.—The prevention of amebic dysentery is not a problem for health officers in this country. It depends upon the same general principles as are necessary in preventing the spread of any disease caused by contamination of food and drink by the intestinal discharges of man.

CHAPTER XXI

HOOKWORM (ANCHYLOSTOMIASIS)

Definition.—Hookworm disease is caused by a small round worm, about three quarters of an inch in length. It is characterized by anemia, malnutrition, palpitation of the heart, bodily weakness, and disinclination to exertion. The disease appears to retard children mentally, and the disinclination to exertion has often caused a false diagnosis of laziness or stupidity.

History.—The seriousness of hookworm disease and the anemia it caused was first appreciated during the building of the St. Gothard Tunnel in 1879. Perroncito showed that the disease was due to hookworms. Hookworm disease is common in tropical and subtropical countries; and occurs in the colder climates only in mines or tunnels, such as those of Germany, Wales, Belgium, France, and Spain.

In this country, it has caused enormous economic loss in the Southern States; and is still a common infection among the poorer classes. Hookworms were doubtless introduced by the negro slaves. The colored race in America serves as the reservoirs and distributers of the infection. The negroes have a considerable immunity and show very few symptoms compared to the whites. Hookworm disease is serious also because, by lowering resistance, it predisposes to tuberculosis and other diseases.

Stiles, of the Public Health Service, made one of the great contributions to preventive medicine, when he awakened the people of the Southern States to the seriousness of the hookworm. In 1902, Stiles showed that the American hookworm differed from the old world variety; and he named it *necator americanus*.

How the Larvæ Enter the Body.—The larvæ bore through the skin into lymphatics or veins, and go by the right heart to the lungs. They pass from the lungs, through bronchi and trachea to the throat. They pass down the esophagus to the stomach. The larvæ moult several times but become adult forms in about four weeks, in the upper part of the small intestine. Here conjugation of male and female takes place. The eggs are passed in the intestinal discharges. The adult worms attach themselves to the mucous lining of the intestine. The eggs in human feces need moisture, warmth, and shade for development into the larval form which penetrates the human skin to reach the intestine as described above.

The eggs often find favorable conditions in a porous sandy soil in a warm climate. Infection is common in people who go about barefooted. The feet often show an inflammation of the skin (dermatitis), called "ground itch." This is caused by the feet coming in contact with feces, polluted soil containing the larvæ, which irritate the skin in passing through. While the great bulk of infections takes place through the feet, the larvæ may pass through the skin in any other location. There are other possibilities of infection: the larvæ may enter the body through water or food, or from contaminated fingers or objects.

Control.—The prevention of hookworm is relatively simple. It necessitates proper sewage disposal. It is pre-

eminently a disease of soil pollution, and when human excreta is properly cared for, hookworm disappears. It naturally disappears from cities, as the sewerage system is extended; but equally good results have been attained in rural districts by the installation of sanitary privies. Hookworm patients are treated by thymol, and other worm medicines, to rid them of the worms.

PART FOUR

DISEASES SPREAD BY INSECTS

CHAPTER XXII

MALARIA

Definition.—Malaria is a febrile disease, caused by a protozoal parasite, the plasmodium of malaria. There are three species of plasmodium, each responsible for a different type of the disease: *Plasmodium vivax* causes a benign "tertian" variety of fever, or a paroxysm "chill and fever" every alternate day. *Plasmodium malariæ* causes a quartan variety, or a paroxysm every seventy-two hours. *Plasmodium falciparum* causes the malignant tertian or estivo-autumnal fever. This is the pernicious malaria of the tropics.

In the first two benign types, tertian and quartan, we have clearly defined cold, hot, and sweating stages. The paroxysm begins with a pronounced chill, followed by fever and subsidence of the fever with profuse sweating. In the malignant tertian, the chill is less pronounced—the so-called "dumb" chill with a prolonged hot stage. When these parasites of malignant tertian accumulate in great numbers in the blood vessels of the brain, we have the "pernicious malaria" with marked cerebral symptoms, coma, and death.

History.—Malaria was described by Hippocrates, and Varro attributed it to the miasma arising from the marshes. Cinchona or peruvian bark was introduced into Europe about the middle of the seventeenth century.

In 1880, Laveran, a French army surgeon, noted and

described the malaria parasites in human blood. The Italian Golgi contributed the fact that the malarial paroxysm was coincident with the simultaneous rupture of numerous sporulating forms (merocytes) in the blood. Golgi, with his contemporaries Marchiafava and Celli, showed the different types of malaria due to different species of plasmodia.

Improved staining methods (Romanowsky) made possible a close study of the changes in the parasite during the cycles. Manson, in 1894, formulated an hypothesis of the mosquito transmission of malaria, and Ross, Grassi, and Bignami followed out the mosquito cycle of development. In 1900, Sambon and Low, living in a screened hut in the Roman Campagna, demonstrated that protection against bites of mosquitoes prevented malaria. Later, infected mosquitoes, shipped from Italy were allowed to bite volunteers in England, who promptly came down with malaria.

Prevalence.—Fifty years ago malaria was common in the Middle West and Plains States. The northern boundary of the endemic area has receded year by year, until to-day it is a serious problem in no state north of the Ohio River. It seems to find the warm climate most favorable and tends to disappear from the colder areas first. While mortality statistics fail to show the damage done by malaria, they are useful in showing the relative prevalence in the various states (see table on page 197).

Alabama, Georgia, Texas, and Arkansas are not in the registration area, but must be included among the states in which malaria is a serious problem.

It is difficult to estimate the seriousness of malaria from mortality statistics, because in the United States the disease does not have a high mortality. Mortality statistics alone do not give proper weight to malaria as a public health

DEATH RATE PER 100,000 POPULATION

MALARIA 1920

State	*Death rate per 100,000*
Mississippi	38.2
Florida	35.1
Louisiana	32.5
South Carolina	28.8
North Carolina	8.1
Tennessee	7.8
Missouri	4.
Kentucky	2.5
Virginia	2.2

problem, because they fail to show the tremendous economic loss due to disabling sickness of malaria. Deaths from pneumonia, typhoid, or tuberculosis are recorded as such. Malaria disables, cuts down efficiency, and prepares for death, which is usually recorded as due to some other terminal affection. Carter of the Public Health Service, our greatest authority, has this to say of the seriousness of malaria as a problem:

The hot countries are pre-eminently the home of protozoal infections, and in the southern parts of the United States one such disease, malaria, stands foremost for the injury it does. In that section not one of the bacterial diseases is in its class in this respect, not even excepting tuberculosis. And here, let me say, that in making this statement, I am only considering such parts of the South (and Southwest) in which malaria prevails *to such an extent as to create a serious sanitary problem.* In many sections of the South it is no problem at all; in many others it is a very minor problem; but in those sections where it is really prevalent, the question of malaria easily constitutes the most important sanitary problem with which we have to deal. There it stands first on the list for the injury which it does the community.

The recorded mortality of a disease frequently does not indicate its true influence on the earth rate. This is eminently true of malaria. From its effects, physical and economic, in lowering the general vitality of a community, it is a causal factor in many a death in which it is not the *terminal* factor, the one recorded as the "cause of death." *Mortality statistics do not, then, give the proper weight of this disease as a cause of death.*

It is not in its death rate that the gravest injury of malaria lies: It is in its sickness rate, in the loss of efficiency it causes, rather than in the loss of life. One death from pneumonia ordinarily corresponds to about 125 sick days—work days lost; one from typhoid fever to 450 to 500 sick days; one from tuberculosis to somewhat more than this among whites, decidedly less among negroes. A death from malaria, however, corresponds to from 2000 to 4000 sick days. This loss of efficiency may really be doubled or trebled, for the man infected with malaria is frequently half sick all the time.

And it is the *amount* of *malaria* when it is bad which appalls. If one per cent of the population is stricken with typhoid fever, it is an epidemic and a bad one. Contrast this with 40 per cent to 60 per cent of a population per annum affected with malaria, and I have seen outbreaks with 90 per cent, and you gain some idea of the importance of this disease. The loss of efficiency caused by malaria in the country of the malarious section of the South is beyond comparison greater than that caused by any other disease, or even by any two or three diseases combined, including typhoid fever and tuberculosis.

Cause.—The plasmodia of malaria are small protozoal parasites which have the power to enter the red cells of the blood. They possess ameboid movement, produce pigment, extrude whiplike processes, and have two distinct methods of reproduction: one, sexual and the other, non-sexual. The form of the parasite injected into man by the mosquito is called the sporozoite. The reproduction in the human body from the sporozoite is non-sexual. The sporozoite enters

a red cell, enlarges and becomes round. This large body (merocyte) divides into a number of spore-like bodies, and enlarges until it occupies almost the entire space within the red blood cell. It then bursts and frees into the blood stream the new crop of parasites; these attack other red cells and the process is repeated.

When parasites (sporozoites) are first introduced into the blood by a mosquito, they are too few to cause symptoms. They rapidly increase and in about two weeks symptoms may be expected. These symptoms are due to the rupture at about the same time of a large number of merocytes, and the liberation of a large amount of toxin produced by the action of the parasites in the red blood cells. This non-sexual cycle in man is called schizogony. In addition to schizogony, a non-sexual reproduction, the parasites in human blood develop sexual forms called gametes. The female gamete has more pigment but less chromatin and is called a macrogametocyte; the male gamete with less pigment and more chromatin is called a microgametocyte.

These gametes, male and female, have no further development, unless the blood containing them is withdrawn from the human body.

Sexual Cycle in the Mosquito.—When an anopheles mosquito bites a malarial patient, these gametes undergo a sexual phase in the body of the mosquito. The male microgametocyte gives off whip-like particles which have an active lashing movement. These fuse with and fertilize the female gamete to form the zygote. By a worm-like motion the zygote bores through the walls of the mosquito's stomach, and comes to rest, resembling a cyst. It enlarges rapidly and after a week is full of slender bodies—sporozoites. The mature zygote ruptures its capsule about the tenth day. The

sporozoites free in the body cavity make their way to the salivary glands of the mosquito, and are from there introduced into the human body by the bite of the mosquito. This sexual cycle in the mosquito is called sporogony and takes ten or twelve days. The sexual cycle of the parasite (sporogony) takes place in the mosquito, and this fact marks the mosquito as the definitive host. Man is an intermediate host in whom non-sexual reproduction (schizogony) takes place.

Source.—The only source of human malaria is man himself. There are parasites resembling plasmodia found in birds, monkeys, bats, and squirrels; but these are distinct from the plasmodium which causes malaria in man and are not transmissible to man.

Transmission.—Malaria can be transmitted from man to man only by the mosquito, and only by certain species of anopheles. It is the female that bites, serves as host to the parasite, and infects man. The male anopheles are seldom seen except near the hatching place and are very shortlived. There is relatively little immunity to malaria, although the parasites may be too few to cause symptoms; under these conditions, we have latent malaria, in which an acute attack may be precipitated by exposure, fatigue, or excesses. This latency explains the appearance of symptoms in a location where mosquitoes are absent.

Carter, in his terse, forceful way thus summarizes the transmission of malaria:

> Without going into the question of the conveyance of malaria by the mosquitoes, I will lay down a few postulates:
>
> 1. Malaria is caused by parasites in the blood of the person suffering from it. Persons with such parasites in their blood are infected with malaria.

2. Those parasites were injected into the person by the bite of a mosquito infected with the parasite. Man receives infection in no other way.

3. The mosquito herself received this infection by having previously fed on a person whose blood contained such parasites. The mosquito acquires infection in no other way.

4. The only mosquitoes which are infected with malaria are those of the genus anopheles, and not all species of anopheles are efficient carriers of malaria.

The change from man to the mosquito and back again is necessary for the continuous existence of the parasites, just as necessary as that change for the germ of wheat by which it is alternately in the ground and in the air. The malaria parasite cannot live indefinitely in the mosquito; it cannot live indefinitely, although much longer, in man. Without this continued change between the two hosts the parasite dies. This, then, gives us our clues for malaria control: (1) Keep infected mosquitoes away from man; or (2) keep mosquitoes away from infected men. The control of either host—the mosquito or the man—will eliminate malaria.

Mitzmain of the Public Health Service demonstrated that the parasite dies out in hibernating mosquitoes, and that man is the winter carrier of malaria.

Control.—The cardinal principles of malaria control may be stated as follows:

(1) Eliminate anopheles mosquitoes.
(2) Prevent access of anopheles mosquitoes to man.
(3) Eliminate human carriers.
(4) Protect exposed persons by quinine.

All four of these principles are used in different areas, and all are useful. In the United States, the first is without question the real remedy. There may be situations where prevention of mosquito breeding is impossible because of prohibitive cost, but these are the exceptions. Usually anopheles production can be prevented at a reasonable cost,

considering the economic saving effected, and should be tried first.

The demonstrations made during the Great War by the United States Public Health Service in its work of protecting the health of the military forces in the extra-cantonment areas, have awakened the entire South to the possibilities of control and ultimate elimination of malaria. Some of the camps were located in the most malarious sections of the South, yet, due to the mosquito destruction measures, the sick rate from malaria was negligible. The Public Health Service in making the zones about the camps malaria free, co-operated with the state and local health authorities and depended in greatest measure on controlling the production of anopheles.

The methods employed are draining and filling the breeding places or rendering them unfit for breeding by increasing current, by oiling, by larvicides, or by placing top minnows therein. Carter cites as examples of being worth its cost:

Roanoke Rapids, N. C., is a mill village, or rather a group of mill villages, with a total of over 4000 population. Prior to the malaria work the population was continually changing. Wages were good, work was abundant, and people came, but they developed malaria and would not stay. The mill managers estimated the efficiency of their employees at from 40 to 60 per cent during the four unhealthful months. During this time machines were constantly idle. The mill physicians, who attended employees without charge, averaged during the summer months of 1912 and 1913, fifty calls per day for malaria. During 1914, the first year of malaria work (control of mosquitoes was depended on), there were still a few cases (33) of malaria, relapses from 1913. The efficiency rate rose to 90 or 95 per cent, and the average number of calls for malaria for the same months was three daily. In 1915 there was no question of efficiency to be considered—it was normal. The average of doctors' calls for malaria was one in three days.

All these were on newcomers and were believed to have been contracted elsewhere.

One of the millmen writes: "The money spent in your campaign against malaria here gave the quickest and most enormous returns I have ever known from any investment." It did pay in the first year from 100 to 400 per cent. The cost here was 80 cents per head for the first year and 27 cents per head for the second year. The efficiency of the mill was raised from 50 to 100 per cent (normal).

The second principle mentioned above—prevent access of anopheles mosquitoes to man—while theoretically sound is, according to Carter, not feasible in villages or thickly settled communities where malaria is prevalent. In isolated farm houses and in plantations much can be done by screening. Screening is useful and necessary in preventing the spread from the known case of malaria. The elimination of carriers, the third principle mentioned, is possible if we can find all the carriers and treat them. A few doses of quinine will render the carrier non-infective to the mosquito that bites him, by destroying the parasites in the circulating blood. Carriers are so numerous in malarious areas that the extreme difficulty of finding and treating all of them is obvious.

The fourth principle mentioned: Protect persons exposed to infection by giving continued doses of quinine. So-called immunization by quinine is useful and practiced by many temporary sojourners in the tropics. Carter says:

The use of quinine by well people in small doses to prevent the development of malaria is not admitted to be efficient universally by sanitarians, yet the general consensus of opinion is in its favor, and comparative experiments by the Japanese in Formosa and the Germans in East Africa would show it decidedly valuable. There are two methods of giving it, one from 3 to 5 grains a day and

one a single dose of 10 or even 15 grains once a week. The latter seems to give the best results—certainly in proportion to the quinine taken. The former was the method mainly used on the Canal Zone. It gave, I think, no discomfort to those using it, as a great many of the higher officials and their families did for long periods from 1904 to 1906, when malaria was prevalent. This method, in the opinion of the writer, is mainly for use to tide over a specially bad season. He is sure it prevents sickness; it may not prevent infection.

The subcommittee on medical research of the National Malaria Committee presents the following as a standard method of treatment of malaria for the purpose of curing the patient of his infection and recommends its general use by the medical profession. We believe that this treatment will, in the great majority of cases, prevent relapses in the patients themselves and also prevent transmission of infection to others. Our opinion is based largely upon the results of the treatment by this method, under average conditions, in their homes, of a large number of persons infected with malaria.

For the acute attack 10 grains of quinine sulphate by mouth three times a day for a period of at least three or four days, to be followed by 10 grains every night before retiring for a period of eight weeks. For infected persons not having acute symptoms at the time only the eight weeks' treatment is required.

The proportionate doses for children are: Under 1 year, one-half grain; 1 year, 1 grain; 2 years, 2 grains; 3 and 4 years, 3 grains; 5, 6 and 7 years, 4 grains; 8, 9, and 10 years, 6 grains; 11, 12, 13, and 14 years, 8 grains; 15 years or older, 10 grains.

It is not claimed that this is a perfect or even the best treatment in all cases, but it is our belief that it is a good and satisfactory method for practical use to prevent relapse and transmission to other people.

(Signed) C. C. Bass, *Chairman*; William Krauss; William H. Deaderick; George Dock; Charles F. Craig.

There are drawbacks and limitations to all the measures suggested, and in the solution of the malaria problem in

this country a combination of all is necessary. In industrial centers, mill villages, or thickly populated areas, mosquito destruction work is economically feasible. Malaria, however, is largely a rural disease and the destruction of breeding places to protect a sparse population would, in many instances, carry with it a prohibitive cost. Such situations must depend on protection of the individual by screening, and administration of quinine to eliminate carriers and prevent the development of symptoms.

CHAPTER XXIII

YELLOW FEVER

Definition.—Yellow fever is an acute febrile disease, characterized by rapid rise of temperature with severe headache and backache; and after three days, jaundice, hemorrhages, "black vomit," and suppression of urine. The pulse in yellow fever does not accelerate in proportion to the height of the fever. It is an exception to most febrile diseases which with high fever have a rapid pulse. The pulse in yellow fever is relatively slow.

History.—Yellow fever may have been the disease which played havoc with the second expedition of Columbus in San Domingo, in 1495. It has been claimed, however, that the disease was imported from the West Coast of Africa with slaves. Its origin must be left in dispute. It caused great loss of life in American cities after the notable outbreak in Philadelphia in 1793. It ravaged our Southern ports and spread inland from the Gulf. The outbreak about Memphis (1878) was an appalling disaster. The terror inspired by yellow fever paralyzed commerce, and caused the imposition of barbarous inland "shotgun" quarantines in the closing decades of the nineteenth century.

The general impression before 1900 was that yellow fever was spread by infected "fomites," or articles of clothing, food, or cargo exposed to yellow fever. Nott of Mobile, in 1848, believed in the insect transmission of yellow fever, but

did not single out the mosquito. Finlay in Havana, in 1881, stated that the stegomyia mosquito was responsible for the spread of yellow fever.

In 1898 Carter, of the United States Public Health Service, observed that a period of two weeks elapsed between the first case of yellow fever and the appearance of secondary cases infected from the first. He called this "extrinsic incubation," indicating that the yellow fever virus incubated outside the human body before it was capable of causing secondary cases. The natural inference in view of the work of Manson, Ross, and the Italians in malaria was that this "extrinsic incubation" took place in the body of a mosquito. This observation of Carter's had an influence upon the work of the Army Commission which in 1900 solved the riddle of yellow fever.

The United States Army Commission, Reed, Carroll, Lazear, and Agramonte, went to Cuba and by a series of convincing experiments established the following facts: Fomites had nothing to do with the spread of the disease. A species of mosquito *stegomyia calopus* (now called *ædes* calopus), when allowed to bite a yellow fever patient in the first three days of the disease, became infected and after twelve days (but not before) could transmit yellow fever to a susceptible individual. The infected mosquito continued infectious for its entire life. The Commission also demonstrated that the blood of a yellow fever patient in the first three days of the disease, injected subcutaneously into a susceptible, caused yellow fever and that the filtered blood produced the same results—showing that the cause of yellow fever was a filterable virus.

Virus.—The infectious parasite which causes yellow fever is unidentified, although we know many things con-

cerning its life history. We know it is a filterable virus. The fact that it has two hosts, mosquito and man, suggests that it is probably a protozoal parasite. Recently, Noguchi described a protozoal organism, a spirochete like a spiral thread, which he calls *leptospira icteroides*. He claims that this is the cause of the disease, and that he finds it in all cases. Noguchi's claims, while unconfirmed, are strong, because they accord with the known facts concerning the life history of the parasite.

Prevalence.—Since the epoch-making discoveries of Walter Reed and his associates, yellow fever has lost much of its terror. It has been unable to secure a foothold in the United States since 1905, when a considerable epidemic centering about New Orleans was quickly suppressed by the Public Health Service applying measures directed solely against the stegomyia mosquito. Yellow fever still exists and is reported from the West Coast of Africa, parts of the West Indies, the coast of the Gulf of Mexico, Central America, and South America. Stegomyia mosquitoes are found as far north as Norfolk, Va., and the possibility of yellow fever being introduced exists in any section which produces stegomyia. Close observation is maintained by the Public Health Service through its officers and consuls in foreign ports, and quarantine safeguards at our own ports are regulated according to the prevalence in the infected ports.

Immunity.—The immunity conferred by an attack of yellow fever is strong and lasting. The disease in childhood may be mild and unrecognized, and the large number of immunes in old yellow fever centers is due to these mild attacks in childhood. Carter has shown that the admission of thousands of such "immunes" through quarantines into

the United States is devoid of danger; while a large percentage of non-immunes from the United States, who go to yellow fever territory, contract the disease.

Source.—The source of yellow fever is man in the first three days of the attack, and infected mosquitoes of the genus stegomyia, now called *ædes calopus*. The consensus of opinion is that there are no human carriers of the disease, except patients in the first three days of their illness.

Transmission.—The spread of yellow fever depends entirely on the distribution of the mosquito *ædes calopus*. This mosquito is rarely found at altitudes above 3000 feet. It prefers the coastal plain, between 38 degrees north and 38 degrees south latitude. It is a night feeder, and rarely bites before late afternoon or nightfall. It bites without a warning sound.

The yellow fever mosquito is a house mosquito, and is commonly found in the small collections of water in broken bottles, tin cans, roof gutters, cisterns, or other accumulations of water in the close vicinity of houses. It is a poor traveler, and breeds, lives, and dies within a small radius, frequently one room; it rarely travels seventy-five yards from its breeding place.

A yellow fever mosquito, having bitten a patient with yellow fever, is infected for life, and after twelve days can transmit the disease to a susceptible (non-immune) person; this is the only way in which yellow fever is spread. A yellow fever mosquito after being infected has been known to live 154 days. Life under natural conditions is probably much shorter, but is amply long enough to explain the rôle played by ships in carrying the disease. Fruit and sugar ships furnish favorable conditions for the stegomyia. Infected mosquitoes are probably carried aboard with cargo

or provisions. A case on board may infect mosquitoes bred on the vessel and even if the crew are immune these infected mosquitoes may bite non-immunes at the port of arrival in the United States.

Control.—The control of yellow fever is not difficult, since accurate knowledge is available as to its mode of transmission. The fact that there are no human carriers simplifies control to screening the sick case for three days to avoid infecting mosquitoes; and a direct attack on the breeding places of the mosquitoes. Cases frequently reach our quarantines but are held under screens, and the vessel fumigated to destroy all mosquitoes. Carter showed that vessels anchored in Havana 400 yards off shore, did not become infected unless the crew went ashore.

The destruction of stegomyia mosquitoes in a large city is feasible, as demonstrated by White of the Public Health Service in New Orleans, in 1905. After a very vigorous campaign of several months, it was a difficult matter to find any stegomyia mosquitoes in the city. This was accomplished by screening cisterns, and eliminating all collections of water in the vicinity of houses. The screening of all cases for the first three days would theoretically eliminate yellow fever; unfortunately in an epidemic the mild cases escape observation and control, so that screening of the known cases alone does not suffice, as the mosquitoes become infected from these mild human cases.

Man after his attack is not a carrier, and the really effective measures, after screening of the sick, consist in destroying infected mosquitoes in the houses by fumigation, and eliminating their breeding places in the vicinity of human habitations. The agents used to destroy mosquitoes

are sulphur, hydrocyanic acid gas, pyrethrum, and a combination of phenol and camphor.

In destroying mosquitoes in houses it is best to use pyrethrum (insect powder) or phenol-camphor. These cannot be depended upon to kill mosquitoes, but stun them and they are then easily collected and removed. Sulphur is an effective fumigant, but has limited use in rooms because of its destructive effect on fabrics and metals. Hydrocyanic acid gas does not injure fabrics or metals, and is the surest killer of all insects. It is the fumigant best suited for insect or vermin destruction in ships, warehouses, docks, stables, outhouses, or railroad cars. Its great drawback is its extremely poisonous qualities. It is very effective, but a dangerous procedure in unskilled hands, and its use in houses is limited for this reason.

CHAPTER XXIV

DENGUE AND FILARIASIS

Dengue

Definition.—Dengue or "breakbone" fever is an acute infectious disease characterized by fever, joint and muscle pains, and sometimes an eruption on the skin. It is a common disease in tropical and sub-tropical climates; and occurs in the Southern United States. Dengue is spread by mosquitoes and the evidence is strong that it is spread in no other way. The *culex fatigans* was shown by Ashburn and Craig, in Manila in 1907, to be a transmitter of the disease. They also observed that dengue is due to a filterable virus. The infectious agent has not yet been identified.

Dengue is not a highly fatal disease; it occasions great discomfort and suffering, and the convalescence is often slow. It occurs in widespread epidemics, sometimes coincident with outbreaks of yellow fever. There is no definite immunity following an attack; and a large percentage of the community are often attacked. Dengue attacks with great suddenness, with high fever and pain above and behind the eyes. The fever falls to about normal on the third or fourth day; and after from one to three days rises again abruptly. This gives the characteristic "saddleback" temperature curve. With the second rise of temperature come the rash and the very severe pains in the joints and muscular

attachments. The rash resembles measles, but begins on the hands and feet, sparing the face or involving it last.

History.—The first description of dengue in America was presented by Benjamin Rush after the epidemic in Philadelphia, in 1780. The mosquito transmission of dengue is generally accepted; but that *culex fatigans* is the carrier is not so certain. In Australia, in 1916, experiments with *culex fatigans* failed while infection was secured in four out of seven cases by using stegomyia.

Prevention.—Patients should be isolated in screened rooms, and measures for the destruction of mosquitoes instituted.

Filariasis

For more than thirty years, filariasis has been known to exist in Charleston, S. C. Filariasis is a tropical disease, but Francis, of the Public Health Service, found it present not only in Charleston but in Columbia and Beaufort, S. C., Tampa, Fla., and Mobile, Ala.

Definition.—Francis gives the following description:

> Filariasis of man is a condition characterized by the presence in the blood of microscopic, slender, snakelike, motile microfilariæ (round worms) which have a length about thirty times the diameter of a red blood corpuscle and a width about equal to the diameter of a red blood cell; they have a rounded head and a tapering tail; they are the offspring of adult female parent worms permanently located in some tissue of the body and discharging their young into the circulation. The parent worms in the case of *Filaria bancrofti* are located in the lymphatics and lymph glands of man. . . . *Filaria bancrofti* is found in practically all tropical countries and one endemic focus has been located in the United States—at Charleston, S. C. As the blood of man is the normal

habitat of this microfilaria, it was at first called *Filaria sanguinis hominis.*

A most interesting phenomenon connected with this microfilaria is its night activity and for that reason it has also been called *Filaria nocturna;* the blood of a filarial patient examined during the hours around midnight may show microfilariæ in great numbers, whereas the blood from the same patient examined in the hours around midday may show only one or two parasites. An estimated number of 40 or 50 millions of microfilariæ circulating in the blood may give rise to no symptoms since their size permits them to traverse the capillaries unobstructed. "The healthy, fully formed microfilariæ have, so far as we can tell, no pathogenic properties whatever; the parent worm and the immature products of conception alone are dangerous." (Manson.)

The adult worms may obstruct the thoracic duct or occlude the smaller lymphatic vessels or the lymph glands, resulting in a damming back of the lymph, giving rise to lymph œdema of the undrained area, and due to this increased lymph pressure there results a condition of lymphatic varix or a varicose dilated state of the lymphatic vessels which causes enlargement of the part affected; hence "lymph scrotum," "varicose groin glands" and "varicose axillary glands." If the dilated lymphatic vessels located in the tissues surrounding the kidneys, bladder, testicle or peritoneum become overdistended and rupture, there results a chyluria, chylocele or chylous ascites.

Elephantiasis, although common where *Filaria bancrofti* abounds, is not usually accompanied by microfilariæ in the blood; there are reasons for believing that elephantiasis is caused by plugging of the lymphatic glands with the aborted immature ova of the female adult worm located in the glands. This mechanical obstruction results in great enlargement of the leg scrotum or vulva, or the arm and breast, which is commonly known as elephantiasis.

The adult filariæ are males and females and are found only in the lymphatic system of man. The approximate length of the males is 1½ inches and that of the females is 3½ inches. The number of adult filariæ found in a single individual is small; eight worms would be a large number for one person. Conjugation of

the sexes is essential to multiplication and takes place only within the lymphatic system. Multiplication is brought about by the expulsion of embryos by a gravid female. The number of embryos which one female may discharge is very large—probably at least 1000. The embryos escape from the lymphatic system and circulate in the patient's blood as microfilariæ.

The microfilariæ do not multiply in the blood; any increase in the number of microfilariæ in the blood comes from additional ones thrown into the blood stream from a parent worm located in a lymph gland. The microfilariæ live for a long time in the blood; probably for years. They circulate awaiting the opportunity of being imbibed by a biting mosquito. The mosquito in drawing blood into its stomach draws in also the microfilariæ which, after loosing their sheaths in the mosquito's stomach, pierce the stomach wall and pass to the thoracic muscles where they undergo metamorphosis. After a variable time of approximately two weeks spent in the thoracic muscles, the larvæ migrate to the proboscis and there await an opportunity to get back into man, which opportunity presents itself when the mosquito again bites an individual.

During their stay in the mosquito no multiplication of larvæ takes place. The larvæ when they are ready to leave the proboscis measure one-twenty-fifth to one-sixteenth inch in length.

The larvæ on being delivered back to man by the bite of an infected mosquito pass by way of the lymphatics to the nearest lymph gland where they grow to be adult filariæ.

The cycle, as outlined, starts with the parent worm located in a lymphatic gland giving off her microscopic embryos and ends with those embryos developed into three-inch worms and again located in a lymphatic gland.

Francis explains why the disease does not spread rapidly from the focus in Charleston:

1. No multiplication of microfilaria in the mosquito, and small number of parasites in the mosquito.
2. Sex is important in the few parasites injected by the mosquito.

If male and female are separately located in different glands, conjugation is prevented.

3. Filaria can only be withdrawn from human blood by the mosquito for a few hours before and after midnight.

4. The parasites are not really "injected" by the mosquito, but are deposited on the skin; and must work their own way to the lymphatics.

These and other difficulties explain why a focus established since the days of the slave trade, has not spread to an alarming degree.

Prevention.—The disease is transmitted only by the *culex fatigans* mosquito. Destruction of this mosquito and his breeding places is not difficult, as it breeds in fresh water in back yards, in old tin cans, cisterns, rain barrels, and other accidental containers of water in the vicintiy of human habitation.

CHAPTER XXV

PLAGUE

Definition.—Plague is an acute infectious disease caused by the *bacillus pestis* or plague bacillus, characterized by high fever, stupor, and toxic action on the heart. It is primarily a disease of rats and other rodents, and is transmitted to man by fleas. The common type of the disease is the bubonic, marked by swelling of the glands (bubo) of the groin or axilla. There is a pneumonic form, which is really a pneumonia due to the plague bacillus. There is also a septicæmic form quickly fatal, in which the blood of the patient teems with plague bacilli.

History.—The term plague has been used by ancient writers to designate many different diseases which carried off great numbers of people. Certain of these plagues occurring before the Chirstian era were bubonic plague. There is a description in the Bible of a plague with buboes and accompanied by death of mice in great numbers. The famous "Black Death" of the fourteenth century was undoubtedly plague. It came from China through Asia Minor and Egypt to devastate Europe, where it is estimated one quarter of the population died. This outbreak was responsible for the first official quarantines. The Great Plague of London, in 1665, cost 60,000 lives out of a population of 450,000.

Plague was common in Europe in the eighteenth century,

but by 1840 had disappeared. The present pandemic of plague has spread over two hemispheres. It began in China in 1894, and it is said to have caused in that year 100,000 deaths in Canton. From Canton and Hong Kong, it has slowly spread to India, Egypt, Japan, and the Philippines; thence to Europe, South Africa, Australia, and the United States, Central, and South America. The loss of life in India has been expressed in millions.

Prevalence.—Plague has become a disease of worldwide distribution, but our interest in the United States is centered upon its entrance through our seaports. It obtained a foothold in San Francisco in 1900; the rats of Chinatown were probably infected by rats from vessels in the Oriental trade. Later outbreaks were suppressed by the United States Public Health Service, and San Francisco has been free of plague since 1908. In 1914, an outbreak occurred in New Orleans. In 1919, another outbreak occurred in New Orleans, and in 1920, the disease appeared for the first time in Galveston, Tex., Beaumont, Tex., and Pensacola, Fla. The vigorous measures applied by the United States Public Health Service have in each instance quickly checked the human cases, and after eighteen months seem to have eliminated plague from the rat population.

The geographical distribution of bubonic plague is peculiar. It is true it is worldwide in the sense that it has encircled the globe, yet it has shown no tendency to spread north of latitude 40 except in the Mediterranean and our own Pacific Coast (Seattle). This peculiar distribution suggests strongly that climate is a factor in the spread of plague. The climate of the Pacific States and the Mediterranean is much milder than that of countries farther south in other parts of the world.

Bubonic plague has occurred in England and Scotland (Glasgow), but shows no tendency to spread. It has spared the Scandinavian countries, Canada, and the Atlantic Coast of the United States. Cold climate has a direct effect on flea breeding, and the most logical explanation of the relative immunity of northern cold climates, such as Boston or New York, from bubonic plague is that rats have few and often no fleas during the cold winter months.

Pneumonic plague, on the other hand, is partial to cold climates. Its worst recorded outbreak occurred in Manchuria, a very cold country, above latitude 50. Pneumonic plague is really a pneumonia caused by the plague bacillus. It is not spread by fleas but by the sputum, in exactly the same way as pneumonia.

Cause.—Plague is caused by a rod-shaped organism with rounded ends, a bacillus discovered by Yersin in 1894, in Hong Kong. The bacillus stains with anilin dyes, heavily at the ends, faintly in the middle, giving a "bipolar" staining. In smears from glands or other material, the appearance of somewhat oval bacilli with bipolar staining is suggestive of plague; especially with bubo and clinical signs of plague. Definite diagnosis depends upon inoculation of the suspected material into guinea pigs or rats. These animals are so susceptible to plague, that plague material rubbed on the shaven skin is sufficient to produce plague in the animal. The incubation period of plague is usually from three to seven days.

Source.—The source of plague is the rat, or some other rodent. In America the Californian ground squirrels became infected, and the disease still exists among them. They are not migratory, are rural in habitat, and do not come in close contact with people. On the other hand, the rat is a

great traveler, is urban in habitat, and lives in close association with people. The rat, therefore, is by far the more dangerous source. The ground squirrel is dangerous chiefly because he may reinfect the city rat population, through the intervening suburban rats. In China and Thibet a similar rodent, the marmot, constitutes a reservoir of infection. Under certain insanitary conditions, overcrowding, and cold climate, terrific epidemics of pneumonic plague have occurred in North China.

In the pneumonic form man is the source of the disease, as it differs from other pneumonias only in its cause, the *bacillus pestis,* and in uniform fatality. Pneumonic plague has never occurred in epidemic form in America, although one group of about a dozen cases occurred in Oakland, California, in recent years. The combination of insanitary overcrowding, filth, and climate, which produced the horrible Manchurian epidemic of pneumonic plague, probably does not exist in our country.

Routes of Infection.—The transmission of the pneumonic form is identical with that of pneumonia caused by other germs. It is so far of only academic interest in the United States, as our epidemic experience with plague is confined to the bubonic variety. The rat disease is characterized by the presence of the germs in the rat's blood. Fleas feeding on the rat ingest the bacilli. They seem to cause an obstruction at the lower end of the esophagus, so that when an infected flea attempts to feed again, he is unable to do so; and the contents of his esophagus are regurgitated on the skin. Inoculation of man or rodents takes place in this way. Fleas infected by biting an infected rat leave the rat when he dies and seek another, feeding on other rats or man, or any warm blooded animal they encounter. There is ample

evidence to show that the bubonic form of plague is spread from rat to rat, and from rat or squirrel, to man by the flea, and in no other way.

Immunity.—One attack of plague confers immunity probably for life. Artificial immunity may be produced of either an active or passive character. Active immunity can be produced by the injection of a vaccine (Haffkine's prophylactic) made up of cultures of plague bacilli, killed by heat. It gives a very considerable degree of protection, and a milder form of the disease in the few vaccinated persons who contract the disease. Passive immunity is given for a prief period (ten days) by Yersin's serum. This serum is prepared in the horse by injecting the horse with first killed, and later living, plague bacilli; it is also used in treatment of plague cases and seems to have considerable value. Neither vaccine nor serum are of any value in pneumonic or septicæmic plague.

Control.—The successful eradication of plague in our seaports has been achieved repeatedly by the Public Health Service, by measures directed against the rat alone. The human cases cease in a few weeks, and after about eighteen months, no more cases can be found in rats. Cases of bubonic plague in man are harmless in an environment free from fleas and vermin. They are of interest chiefly as indicating foci of rodent plague where the disease was acquired.

In rat destruction, trapping is the most useful measure, because we have the address, street and number, where infected rodents were caught. Poisons will kill rats, but in an epidemic give no information of the location of infection. Trapping and expert laboratory examination give an index of the course of the epidemic. Trapping does not eliminate the rat population; beyond a certain minimum it does not

reduce it, but persistent trapping gradually eliminates the infection from the rodent population. When either a human or rodent case is discovered, all rat harbors are destroyed; and fumigation of houses, cellars, and outhouses with cyanide or sulphur to kill rats and fleas is indicated. Ratproofing of the infected buildings follows; docks, warehouses, and railroad terminals are ratproofed as rapidly as possible; and special attention is given to slaughterhouses, stables, food and meat markets, and other homes of the rat.

Destroying the customary rat harbors forces the rats into locations where they are easily trapped. Another valuable measure is to cut off the rats' food supply. This is accomplished by ratproofing of food depots and warehouses, and by proper garbage disposal. Failure to protect foods from rats, and improper disposal of the garbage, furnish the rat with ample food and make trapping difficult and ineffective.

CHAPTER XXVI

TYPHUS FEVER

Definition.—Typhus fever is an acute infectious disease, characterized by fever, headache, great prostration, and a peculiar skin eruption. The disease usually begins abruptly with a chill, and the rash appears on the fourth or fifth day. The rash consists in dark red, slightly elevated spots, varying in size from very small to spots ¼ inch in diameter. It appears on the abdomen, flanks, back, and legs, rarely on the face. The spots are darker in the more severe and fatal cases, and hemorrhages in the skin (petechiæ) are common. In the non-fatal cases the fever often terminates abruptly by crisis, about the fourteenth day. In fatal cases death may take place at any time before or during the crisis; or during early convalescence from heart failure.

History.—Typhus fever is one of the great epidemic diseases. It has been called jail fever, ship fever, and camp fever; because it is seen at its worst in conditions of misery, filth, and overcrowding. It has been known for centuries as a highly contagious and extremely fatal epidemic disease in European countries. It took its greatest toll in poverty and filth, and where people were huddled together. Our recently acquired knowledge of its mode of spread, by the louse, explains why this was so. The old English writers describe its ravages, and tell of the prisoners communicating the disease to court officials and even the judges.

It is distinctly a disease of cold or temperate climates. In Mexico, for instance, it does not occur in the hot coastal region; but is confined to the cool plateau at an elevation of not less than 6000 feet. It was formerly confused with typhoid, until Gerhard clearly differentiated the two diseases. We can now understand why typhus fever has declined with the great improvement in sanitation and personal hygiene of the past thirty years. It still persists in Europe and North Africa; and after the Great War was a frightfully fatal scourge in Poland, Central Europe, and the Balkans.

The last epidemics in the United States occurred in Philadelphia in 1883 and New York 1892-3. We have in New York a disease called Brill's disease, which Anderson and Goldberger showed to be typhus fever of a mild character. They also showed that typhus fever of Europe was identical with Mexican typhus called *"tabardillo."*

The disease in New York (Brill's disease) is a smouldering remnant of the epidemic of 1893, and is distinctly milder than the typhus fever of other days. Goldberger, our foremost authority on this disease, says that we must revise our opinion as to its fatality; that although we may have outbreaks with a mortality of from 30 to 50 per cent, it is at times a benign disease with a fatality under 10 per cent.

Typhus is occasionally reported from other large American cities, such as Baltimore, Boston, Philadelphia, Atlanta, Chicago, and Milwaukee. Since the discovery that the disease could spread only through lice, it is no longer seriously regarded as an epidemic problem in the United States.

Transmission.—Typhus fever has for a source, man alone; and is transmitted from man to man by the louse. In 1909, Nicolle in Tunis succeeded in transmitting the disease by the body louse. This finding was quickly confirmed

and firmly established by the work of Ricketts and Wilder, and Goldberger and Anderson in Mexico City. Goldberger and Anderson showed the possibility of transmission by the head louse also. The virus is in the patient's blood throughout the febrile period, and the louse probably may become infected at any time within this period, or possibly three or four days later during defervescence.

The discovery of the part played by the louse has cleared up all the obscure and mysterious features of the disease. We now understand why it is known as the disease of the vagabond and jail-bird, and why it was always associated with squalor and wretchedness. It has not yet been definitely established how soon after biting the louse becomes infective; nor how long he remains so.

Control.—The prevention of typhus fever has been greatly simplified and placed on a sound basis by the discovery of the transmission by lice. The case of typhus fever clean and free from lice, in a room and bed free from lice, is no more dangerous than a case of yellow fever or malaria in the absence of mosquitoes. Goldberger and Neill say:

> We may say, therefore, that to prevent infection of the individual it is necessary for him only to avoid being bitten by the louse. In theory this may readily be done, for we know that the body louse infests and attaches itself almost entirely to the body linen, and that boiling kills this insect and its eggs. Individual prophylaxis is based essentially, therefore, on the avoidance of contact with individuals likely to harbor lice. Practically, however, this is not always as easy as it may seem, especially under the conditions of such intimate association as is imposed by urban life. Particularly is this the case in places, such as some of the large Mexican cities, where a large proportion of the population harbors this vermin. Under such circumstances it will be well to avoid crowds or

crowded places, such as public markets, crowded streets, or public assemblies at which the "peon" gathers.

Community prophylaxis efficiently and intelligently carried out is, from a certain point of view, probably easier and more effective in protecting the individual than is the individual's own effort to guard himself. Typhus emphasizes, perhaps better than any other disease, the fact that, fundamentally, sanitation and health are economic problems. In proportion as the economic condition of the masses has improved—that is, in proportion as they could afford to keep clean—this notorious filth disease has decreased or disappeared. In localities where it still prevails, its further reduction or complete eradication waits on a further improvement in, or extension of, the improved economic status of those afflicted. Economic evolution is a very slow process, and, while doing what we can to hasten it, we must take such precautions as existing conditions permit, looking to a reduction in or complete eradication of the disease.

When possible, public bath houses and public wash houses, where the poor may bathe and do their washing at a minimum or without cost, should be provided. Similar provision should be made in military and construction camps. Troops in the field should be given the opportunity as frequently as possible to wash and *scald* or *boil* their body linen.

Lodging houses, cheap boarding houses, night shelters, hospitals, jails, and prisons, are important factors in the spread and frequently constitute foci of the disease. They should receive rigid sanitary supervision, including the enforcement of measures to free all inmates of lice on admission to institutions.

Destruction of Lice and Nits.—Clipping the hair is advisable but often impracticable. Pierce of the Public Health Service recommends kerosene soap, made by boiling one part soap and four parts of water, and adding two parts of kerosene, followed by shower baths as effective for washing the body. Head lice may be destroyed by tincture of larkspur or equal parts of kerosene and vinegar applied to the

hair, by means of a towel for half an hour (Pierce). Delousing of clothing or baggage is effected by moist heat or steam under pressure. Sterilization in large steam chambers is very effective. First produce a vacuum of 10 to 15 inches, then maintain 20 lbs. for ten minutes (temperature 259° F.).

The practical use of steam at 100° C. (212° F.) is illustrated in a simple form by the "Serbian barrel," which was widely used during the war. Nuttall (1918) states that "a 60-gallon barrel is capable of coping with four soldiers' kits or seven blankets at a time. The barrel having its top and bottom knocked out is provided with a grated wooden bottom and a flat wooden lid which can be weighted if necessary with stones." The barrel is stood upright and used in one of the following ways: (*a*) The barrel is set above a circular boiler, and a tight joint is made so that all the steam passes into the barrel. (*b*) A battery of barrels is set up over the circular boilers, secured in a long, narrow, brick furnace. (*c*) Steam which is led by a pipe to the top of a barrel (with an impervious bottom) percolates downward, and the condensed water is drawn off by a spigot at the bottom of the barrel. Lelan (1917) exposes clothes to be disinfected to thirty minutes' steaming in the Serbian barrel; whereas Lloyd (1919) advises one hour's steaming.

Dry heat is also useful in delousing clothing. The clothing should be loosely hung in chambers which have flues or fans to ensure circulation; steam is preferable in civilian practice. Sulphur is effective in destroying lice, but Nuttall questions its efficacy on nits. Cyanide is effective in expert hands, but it is extremely poisonous and this danger excludes it from ordinary use.

CHAPTER XXVII

ROCKY MOUNTAIN SPOTTED FEVER

Definition.—Rocky Mountain spotted fever is an acute febrile infectious disease, with continued fever, severe joint and muscular pains. There is a petechial rash beginning on ankles, wrists, and forehead, and spreading all over the body. It occurs in the summer months, and has in some sections a very high mortality. There is a striking difference in the mortality in different states. In Idaho, case fatality is as low as 4 per cent, while in the Bitter Root Valley, Montana, it has been as high as 75 per cent. The reason for this variation in severity still remains an unsolved problem. On account of its persistent seasonal prevalence and severity, the disease has become a very serious public health and economic problem in the Bitter Root Valley, where, in addition to the lives that are annually sacrificed, very valuable agricultural lands have depreciated in value and in certain localities have been almost abandoned, on account of the fear and dread that the inhabitants have of Rocky Mountain spotted fever.

History.—The disease has been known in Idaho and Montana since 1873. It has been seriously studied for the past twenty years. In 1902, Wilson and Chowning suggested that the ground squirrel was the host and that the tick *(Dermacentor andersoni)* was the transmitter. Investigations were made by Ashburn, Craig, and Kieffer of the

Army, and by Cobb, Anderson, Stiles, Francis, King, McClintic, Rucker and Fricks of the Public Health Service. McClintic in his work laid the foundation for successful preventive work, but in August 1912 contracted the disease and died.

Prevalence.—The disease has been reported from California, Colorado, Idaho, Montana, Nevada, Oregon, Utah, Washington, and Wyoming.

The Virus.—Spotted fever is caused by a filterable virus, though the parasite has not been described or definitely identified; numerous claims have been made, but all remain unconfirmed by convincing experiment. The virus is transmitted from animal to animal, and to man by the infected wood tick; and in no other way. The transmission is not mechanical. The tick becomes infected and remains so, and the female tick transmits the virus to her progeny.

Prevention.—All measures employed for the eradication of Rocky Mountain spotted fever from the Bitter Root Valley have been directed entirely toward the destruction of the wood tick, *Dermacentor andersoni.* These comprise: (1) The reclamation and cultivation of arable land. (2) The burning over of the foothills. (3) The killing of wild animals. (4) Hand picking and the dipping of domestic animals in arsenical dips. (5) Sheep grazing.

The placing of new land under cultivation on the west side of the valley continues slowly, and, as a rule, by means of small isolated fields surrounded by open, uncultivated land. The full benefit from cultivation as a means of tick destruction is not obtained except in the case of large tracts, and the west-side foothills being too broken for extensive contiguous cultivation, it is not expected that this measure alone

can greatly affect the problem of tick eradication in the valley.

The Forest Service continued in 1914 the systematic attempt to burn over the west-side foothills during the early spring months, while the mountain forests were still protected by snow, but without great success. Frequent rains prevented extensive burning until the snow had disappeared, and the burning had then to be abandoned because of the danger to standing timber. Extensive burning each spring should reduce the tick infestation, but it seems very difficult of accomplishment under existing conditions in the Bitter Root Valley.

The investigation of sheep grazing as a means of tick eradication in the Bitter Root Valley was begun by the Public Health Service in 1913 and reported by Fricks at that time.

The four factors upon which the success of sheep grazing as a tick-eradicative measure depends were given as follows: (1) The removal of undergrowth and the consequent destruction of "good tick country" by close grazing. (2) The destruction or removal from the sheep range of other large mammals, domestic and wild, which serve as hosts for the adult ticks. (3) The destruction of the ticks themselves, principally by means of the lanolin in the wool of the grazing sheep. (4) The placing of the problem of tick eradication on an economic basis, so that it may be carried out on an extensive scale without cost to the government, the state, or the inhabitants of the valley.

It appears that the other measures of tick eradication employed in the valley—cultivation, burning, dipping, and killing of wild animals—have not been sufficiently extensive and either cannot be made so, or, if possible, only at a very

great expenditure of labor and money. These objections cannot be raised against sheep grazing, because once the industry is established on the west side of the valley it will run itself up to the limit of grazing capacity with at least a small profit to the sheep owners. The only question to be considered in regard to sheep grazing as a means of tick eradication is that of efficiency.

The effect of these measures is slowly apparent, but yearly more in evidence. The adoption of proper grazing methods, including legislation restricting the grazing of these badly infected areas to sheep, will gradually eliminate spotted fever. The dipping of domestic animals, while useful, would never of itself accomplish this, but the extermination of rodent animals should be vigorously prosecuted.

PART FIVE

DISEASES DERIVED FROM ANIMALS

CHAPTER XXVIII

RABIES

Definition.—Rabies or hydrophobia is an acute infectious disease of animals, which is transmitted to man by the bite of a rabid animal. All animals are susceptible; wolves, jackals, hyenas, and foxes are frequently infected. It is most common in dogs, and man usually contracts the disease by being bitten by a dog with rabies. Cats also are susceptible, and the bite of a rabid cat has caused the disease in man.

The distribution of rabies is worldwide, although it has been eliminated in Norway, Sweden, Denmark, and England. Rabies is a very old disease. Aristotle in the fourth century B.C. mentions it; and Celsus (first century A.D.) gave a detailed description of it. There were forty-one deaths from rabies in the registration area in 1920, cases occurring in sixteen States.

The cause of rabies is not positively known. In the brain of rabid animals, peculiar protozoa-like bodies, called "negri bodies" have been described and are believed by some to be the cause. Whether they are the cause or not, they are useful in diagnosis, as they are not found in any other disease. The virus is contained in the saliva of the rabid animal, and infection usually takes place through bites or scratches.

Transmission.—A dog infected with rabies can transmit the infection by biting before he shows symptoms. A rabid

dog will show symptoms within fifteen days, so that a dog that has bitten a person and shows no symptoms for three weeks is certainly not rabid and his bite is harmless. The production of the disease in man, and the time elapsing before symptoms appear, depend upon the location of the bite and the amount of virus introduced. Bites through the clothing in parts of the body where the nerves are few and the skin thick, may not produce the disease or it will develop after a long period of incubation. On the other hand, where there are deep lacerated wounds of the hands and face, where nerves are reached and the protective influence of clothing is absent, the disease develops more rapidly.

It must be borne in mind that the disease can be contracted from a rabid animal licking the hand or face, provided there is a break in the skin. While this occurs rarely, it is good practice to avoid sick dogs, and not allow them to come in close contact with children.

Incubation.—The period of incubation is variable. It may be as long as several months, or as short as fourteen days. The virus has a special affinity for nerve tissue, and it proceeds to the brain along the nerve paths. This explains the long incubation and the great variation, according to the location of the bite or point of inoculation.

Diagnosis.—The first symptoms are difficult breathing and inability to swallow. There are convulsions and, later, paralysis and death. The symptoms and inevitably fatal ending, are almost unmistakable; but positive diagnosis can be made only by laboratory methods. The finding of the negri bodies in the brain makes possible a positive diagnosis.

Control.—The prevention of rabies is effected by the administration of what is known as the Pasteur treatment of the bitten person. Pasteur developed a treatment on the

principle of a weakened or attenuated virus. He passed the virus through a series of rabbits, and brought its virulence to the maximum. As the virulence increased, the incubation in the animals became shorter, until, after passage through fifty rabbits, it became uniform and "fixed" at six or seven days; and is known as "fixed" virus. This is very much shorter than the incubation period when "street" virus (the virus from a mad dog) is first used.

Pasteur used the fixed virus because the dose could be accurately judged and regulated. The rabbit spinal cords are suspended in sterile bottles, closely stoppered, and are dried by the action of caustic potash in the bottom of the bottle. The dose is regulated by the number of days' drying. The first injection is made with the cord which has been drying the maximum number of days. Each succeeding day an injection is made with a cord dried one day less.

The practice at the Hygiene Laboratory, Public Health Service, Washington, D. C., is as follows: The first inoculation is with an eight-day cord; the second day a seven-day cord; the third day a six-day cord; so that by the eighth day a cord only slightly attenuated (one day drying) is used.

The long incubation period, the time elapsing between the bite and the development of symptoms, permits us to establish an immunity to rabies by the Pasteur treatment before the time when the symptoms would normally have occurred. Immunity is produced about two weeks after the treatment. In most cases, the time available for immunization is ample; but in cases severely bitten about the face, the incubation period may be less than the time necessary to immunize with the Pasteur inoculations.

The time factor is the determining one, and a loss of several days or a week in starting treatment may be a fatal

delay. The treatment should be instituted at once, without waiting for a positive diagnosis on the dog. The disease is so horrible in its manifestations and so certainly fatal, that there should be no hesitancy in submitting to the discomfort of treatment and incurring the very slight risk. There is inevitably some loss of time, at best, in securing the material for inoculation; but report with request for treatment should be made immediately to the local health department or the State Department of Health.

CHAPTER XXIX

ANTHRAX

Definition.—Anthrax is an acute infectious disease of cattle, sheep, and horses, that is often communicated to man. Anthrax in animals has a worldwide distribution, although it is not common among American animals. Human anthrax is usually caused by handling hides, wool, hair, or other animal products. If the infection enters through the skin, a malignant pustule is produced. If the infection is inhaled with dust, the "woolsorters' disease," a pneumonia-like disease, results. There is also an intestinal infection due to insufficiently cooked meat. Recently a considerable number of cases have occurred in the United States, caused by infected shaving brushes made from horsehair.

Cause.—Anthrax is caused by the bacillus of anthrax, a spore-bearing organism of considerable resistance. The spores resist the moderate action of heat, drying, sunlight, and disinfectants which destroy non-spore-bearing bacteria. Like tetanus, anthrax spores can live almost indefinitely in soil or upon animal products, hides, horsehair, or wool; and germinate and grow when they reach a suitable medium, such as the human body.

Pasteur developed a vaccine against anthrax on the principle of attenuation of living germs, more than forty years ago. The vaccine is effective in immunizing animals, but is not applicable to man. The prevention of anthrax in man

must be accomplished by other means, by preventing the spread of the disease in animals, and by disinfection of animal products. In factories where wool and hair are handled, proper ventilation and measures to take care of dust must be instituted.

The prevention of anthrax in the United States depends largely upon the disinfection of hides and hair. The use of horsehair in shaving brushes should be prohibited. Horsehair for other uses should be disinfected by boiling. Hides can be disinfected in preliminary processes of tanning.

CHAPTER XXX

TETANUS

Definition.—Human tetanus or lockjaw is an infectious disease, characterized by spasms of the muscles of the jaw and neck. It is usually a sequel of lacerated or punctured wounds, such as are produced by toy pistols, giant firecrackers, or nails. Fourth of July tetanus has been greatly reduced, by the efforts of the American Medical Association for saner celebration of the holiday; and by more careful treatment of wounds, with wider use of antitoxin.

Cause.—Tetanus is caused by the infection of a wound or break in the skin with the bacillus of tetanus. The bacillus of tetanus has certain very positive characteristics. It is an anaërobe, that is, a germ which grows best in media from which air is excluded. It is a spore bearer, and this makes it very resistant in an unfavorable environment. It produces a true toxin, so that, like diphtheria, its damage is done by this toxin; and an antitoxin can be prepared which, administered in time, will nullify the effect of the toxin.

Kitasato, in 1889, first grew the tetanus bacillus in pure culture, by using anaërobic methods. He made a further contribution by demonstrating that in fatal cases in mice, the bacilli are not found in the heart's blood; but that death is caused by the toxin (analogy to diphtheria). This work of Kitasato was followed, in 1890, by the discovery (Behring and Kitasato) of antitoxins for both tetanus and diphtheria,

which was the foundation of the application of serum therapy.

Source.—The natural habitat of the germ is the intestine of herbivorous animals. The soil is infected to a considerable depth by the discharges (manure) of horses and cattle. The germs can be recovered from garden earth, dust of stables (manure), and floor sweepings from houses, barracks, hospitals, and public buildings. Tetanus of the parturient woman and of the newborn child (infection at attachment of umbilical cord) occur, but these forms are much more frequent in the tropics. The germs are so widespread in dust and dirt that there is great danger in any wound which is not clean cut, superficial, and easily cleaned. Patients with deep punctured wounds, and lacerated and contused wounds which are difficult to sterilize, should receive tetanus antitoxin promptly. The incubation period is from six to fourteen days.

Prevalence.—Tetanus is not highly prevalent, nor does it cause many deaths compared with tuberculosis or pneumonia. It is, however, much more serious as a cause of death than rabies or anthrax. There were 1472 deaths from tetanus in the registration area in 1920; 41 deaths from rabies and 39 from anthrax. Because of its symptoms, the disease is regarded by the laity as a thing of horror.

Prevention.—The bacillus of tetanus produces a true toxin, like the diphtheria bacillus; and it is this toxin which produces the symptoms of the disease. There is available an antitoxin produced in horses by injecting them with tetanus toxin, which, if administered early enough, neutralizes the toxin and prevents the symptoms from developing. The tetanus antitoxin is worthless after the symptoms of lockjaw begin; the damage is already done at such a time.

To be of value, it must be given on suspicion, immediately after the infliction of any wound which is probably infected deeply and is difficult to sterilize. The size of the wound is of less importance than its character. Tetanus may develop from punctures from pins, nails, needles, splinters, insect bites, or vaccinations—depending entirely on the presence of tetanus germs on the skin or upon the agent which punctures the skin.

It is very unlikely to follow a shallow, clean-cut wound which bleeds freely. All wounds should be thoroughly cleansed. Punctured or lacerated wounds should be freely opened to permit of cleansing and thorough disinfection. The treatment of the parturient woman and the tying and treatment of the umbilical cord in the newborn, should be given under strictly aseptic conditions.

After all, it is safer to administer tetanus antitoxin at the first dressing of all suspicious, dirt-infected wounds. If suspicious of tetanus, 3000 to 5000 units should be given slowly by gravity into the spinal cord, preferably under an anesthetic. The time factor is the important thing as in rabies. A small dose early, 1500 units, prevents symptoms; but after definite symptoms develop, even the largest doses are likely to be of little value.

CHAPTER XXXI

GLANDERS

Definition.—Glanders, sometimes called farcy, is a widespread infectious disease of horses and mules, which is readily communicated to man. Goats, cats, and laboratory animals are susceptible, but cattle are immune. It is a very fatal disease in man and horses.

Cause.—Glanders is caused by a bacillus (*bacillus mallei*). It does not produce spores and is easily killed. In the horse the germs cause inflammatory processes in the mucous membrane of the nose, and are discharged by the secretions of the nose and mouth. In the skin of the horse farcy "buttons" (nodules) form, which break down and discharge, and these discharges may either infect man directly through the hands or be transferred in handling harness, or any of the stable equipment.

Glanders in horses is difficult to control because of the mild unrecognized cases. The frank cases are easily diagnosed, but the use of the laboratory is the only way to make a positive diagnosis. The Mallein test, similar to the tuberculosis test for cattle, shows when animals are free from glanders by giving no reaction. It is not so reliable as tuberculin, however, and the ophthalmic test is simpler and more reliable. In the ophthalmic test, a few drops of concentrated mallein is placed in the horse's eye; a positive

reaction is shown by swelling and inflammation of the lids, and discharge from the eye.

Control.—The prevention of glanders in man depends upon its recognition and suppression in horses. When found, all animals should be tested, and if found positive should be destroyed. The stables should receive a thorough disinfection, including all equipment which could have been soiled with the discharges.

CHAPTER XXXII

MALTA FEVER

Definition.—Malta fever is an acute infectious disease, characterized by fever of an "undulant" type, profuse perspiration, joint pains, and constipation. It has a low fatality, but the fever is long drawn out and convalescence slow. It is endemic in the Mediterranean and is sometimes called Mediterranean fever. It was not supposed to exist in the United States until Craig, U. S. Army, reported the disease in 1905. In 1911, Ferenbaugh, U. S. Army, reported cases in Texas. In 1912, Yount and Looney reported five cases of Malta fever in persons connected with the goat industry, near Wagoner, Arizona. In 1922, an outbreak of more than thirty cases was investigated by Lake of the U. S. Public Health Service in Phoenix, Arizona. This outbreak resulted from the sale of goat's milk, while previous cases reported had been in persons in the goat-raising industry. Lake concludes that Malta fever has existed in the United States for many years, and is endemic in Texas and other Southwestern States.

Cause.—Malta fever is caused by a small germ, the *micrococcus melitensis.* It is a frail organism, easily destroyed by heat, 60 degrees C., for twenty minutes. It has little resistance to disinfectants, but resists drying remarkably well.

Source and Transmission.—It is primarily a disease of

goats, and this is the chief source of the disease. The goats excrete the germs in their milk and urine. Infection of herders and milkers can take place direct, but those not in contact with goats usually get their infection through goat's milk. Accidental infection of laboratory workers occurs and one fatal case has been recorded.

Lake says:

From the investigations of Gentry and Ferenbaugh, Malta fever has existed in Texas for at least thirty-six years, and, from our own, it has existed in Arizona for at least fourteen years. Tests made on goats in both states have shown an infection of representative herds of nearly 20 per cent. The same subtropical climate, together with numerous goat herds, exists from Texas to the Pacific Coast. It is well known that goats are shipped, or are driven, back and forth between the Southwestern States and into other states as well. Whether infected goats will continue to have Malta fever and spread the same when shipped from our Southern States with subtropical climates to Northern States has not been investigated; but from the researches of Mohler and Hart it is altogether probable that they will. With these conditions present, we are convinced that Malta fever must exist in New Mexico and California. There is no doubt that it exists in Mexico; at least one case developing in Mexico was studied by Woolsey in St. Louis in 1918.

Goat's milk is being sold at the present time in a number of cities in the United States, usually as a fancy article and at a high price. It is consumed largely by infants and invalids, particularly by patients with tuberculosis, and in these persons Malta fever could exist for a long time without being recognized. Some of the Phoenix cases were patients with quiescent tuberculosis, fever-free for months. These cases, with a Malta-fever infection superimposed, behaved as cases in which tuberculosis had suddenly become active, and, in the absence of an epidemic, would never have been diagnosed without the most careful observation. The difficulty in recognizing so insidious a disease in infants needs no comment.

More than thirty authentic cases of Malta fever occurred in the

recent outbreak at Phoenix, Ariz., concerning which the following can be stated:

(*a*) These cases were typical clinically and serologically; and in two cases *B. melitensis* was recovered.

(*b*) Goat's milk is known to have been used in all but three of the cases and cannot be excluded in those three.

(*c*) Tests made on the goats supplying this milk gave 18.3 per cent positives for Malta fever.

(*d*) The disease remained unrecognized for at least six weeks, under conditions unusually favorable for its recognition.

It is extremely difficult to recognize Malta fever early in the course of an outbreak, from the clinical symptoms alone; usually the possibility of its presence is not considered, and laboratory tests are not made for some weeks. The prohibition of the sale of goat's milk in cities where an ample supply of cow's milk is available should be seriously considered by health officers. Where it is necessary to allow the sale of goat's milk to secure sufficient fresh milk, efficient pasteurization under constant supervision by the health authorities should be required.

CHAPTER XXXIII

TRICHINOSIS

Definition.—Trichinosis is a disease caused by a round worm, known as trichina (*trichinella spiralis*) which passes through its complete life cycle in animals or man. Man contracts the disease by eating underdone pork. The larvæ imbedded in the hog muscle (pork), if not destroyed by cooking, are set free in the human stomach; they enter the intestine and in a few days become mature adult worms. The female produces hundreds of embryo worms, and these penetrate the bowel, and are carried by the blood current to the muscles, when they come to rest and encyst themselves.

The common host of the trichina is the rat, which becomes infected from the refuse of slaughterhouses and butcher shops. The hogs become infected by eating rats, or from feces and infected offal. Man receives his infection by eating trichinous pork, which has not been cooked sufficiently to kill the encysted larvæ.

Prevalence.—The U. S. Dept. of Agriculture examinations of hogs reveal from 1 to 2 per cent infected in the United States; and about the same percentage of human bodies at autopsy show presence of trichinæ. It is a very common disease in Germany, where it is the custom to eat raw or imperfectly cooked pork, especially in sausage.

Prevention.—The surest prevention of trichinosis is through cooking of pork. A temperature of 160 F. kills the

parasite. Pickling, smoking, and drysalting of pork may and probably usually does kill the trichina; but the only absolute safeguard is thorough cooking. Trichina larvæ are also killed by refrigeration for twenty days, if the temperature is below 5 degrees F. The source in the rat and hog suggest excellent reasons for rat extermination and improved sanitation of slaughterhouses.

PART SIX

MISCELLANEOUS DISEASES

CHAPTER XXXIV

THE VENEREAL DISEASES

The magnitude of the venereal disease problem is so great that a special volume is necessary in order to do justice to the subject. It is not possible to treat the subject comprehensively and exhaustively within the scope of this book. Only a very general outline will be given, and for detail the reader is referred to special volumes upon the venereal diseases.

Conservative writers have placed the venereal diseases as second only to tuberculosis and pneumonia among the great plagues of the human race. Exact figures are not available, but, from reasonable deductions, it seems probable that the venereal diseases really stand first and cause far greater damage to the race than tuberculosis itself.

Until recent years little, if any, notice of the venereal diseases had been taken by health officials. In 1912 the Health Department of New York City made the venereal diseases reportable—later they were made reportable in California. In 1915 Massachusetts established free Wassermann (blood) examinations for the diagnosis of syphilis, and began the manufacture of arsphenamine or salvarsan (606) in defiance of the German patents, and arranged for free distribution through clinics.

In general, however, there was a complacent acceptance of the horrible toll exacted by these diseases in ruined homes,

childless marriages, blindness, insanity, and invalidism, until the outbreak of the war.

Very active measures were taken upon our entry into the war. The Surgeons-General of the Army, Navy, and Public Health Service, and the Commission on Training Camp Activities co-operated in reducing the hazard to the fighting forces due to venereal diseases. Prostitution and alcoholic beverages were banned from wide zones surrounding the camps, and healthful recreation was provided for the soldiers and sailors.

The U. S. Public Health Service and the Red Cross maintained clinics for treatment in the extra-cantonment zones, to reduce the incidence of the disease in the civilian area to which the soldier had access. The Army and Navy furnished prompt, efficacious, early treatment to those who were infected or had been exposed. All this activity greatly reduced the incidence of the disease in our troops but the outstanding fact was that five sixths of the venereal diseases in the army was contracted in civil live before the recruit entered the service.

Congress recognized the gravity of the situation and an act was passed creating a Division of Venereal Diseases in the Public Health Service. This act also created an interdepartmental Social Hygiene Board to correlate the efforts of the War, Navy, and Treasury Departments. The first appropriation was over four million dollars. A large part of this was apportioned to states to match like sums raised by the states. Practically every state complied with the federal requirements and established a Division of Venereal Disease in the State Departments of Health and made venereal diseases reportable.

Age-old controversies were ended and time-worn fallacies

destroyed by the war. Among the false doctrines which went into the discard were the inevitability of prostitution, the impossibility and harmful influence of continence, and the belief that venereal diseases were uncontrollable because they were secret and "different" from other diseases. The demonstration made in war time taught us that these diseases can be controlled, and that, if health officers do their full duty, the fight must be carried on in peace time with the vigor and efficiency exhibited during the war.

Syphilis

Syphilis is an infectious disease whose manifestations appear in every tissue of the body. Its symptoms are so varied and so inclusive that the great Osler said, "Know syphilis and all other things clinical will be added unto you."

It begins as an initial sore or "chancre"—a small indurated ulcer—which appears about three weeks (not less than ten days) after infection. From the initial sore or "chancre," the germs spread through the body by way of the lymphatics. The glands swell and then the infection becomes generalized. Skin eruptions of varied character and ulcers on the mucous membranes appear and this is known as the second stage.

The third stage comes on after months or years and is characterized by the formation, in great numbers, of a tumorlike growth called a "gumma." The gumma is a poorly organized growth of new tissue, which ulcerates with considerable destruction of tissue, and scar formation.

Syphilis also produces premature old age by changing healthy tissue into scar tissue. These changes from a tumorlike formation (gumma) to ulcer and scar may occur

anywhere in the body, and account for the great variety of symptoms produced. Great damage to the brain, heart, and blood vessels follow, and syphilis causes one hundred per cent of general paresis and locomotor ataxia, and a large percentage of cases of early apoplexy, valvular disease and aneurism.

It is impossible to state the prevalence of syphilis with accuracy. Only recently has it been a reportable disease, and the reporting is very incomplete. Estimates can be made and Vedder estimates that 20 per cent of young men who enlist in the army, and 2 to 5 per cent among the men who enter our colleges, are infected. Collie, in a survey of British working men, found 9 per cent to have syphilis. Among criminals 20 to 40 per cent are syphilitic and from 50 to 100 per cent of prostitutes. Commenting on these figures, Stokes says:

"It needs no comment to carry home the meaning of these figures to every thinking man and woman. Nothing could more effectively shatter the notion that syphilis is the heritage of the unfavored few, the trophy of debauch, the sign manual of the down-and-out. Syphilis is one of the most widespread of all infectious diseases. Its victims are numbered in millions, not in hundreds. Not a man lives, or a woman, who does not elbow it every day, whose house has not seen its entry and departure, who may not at any hour have his name added to the rolls. While, to be sure, there are variations in the nearness or remoteness of the risk, never does it become so distant that any one of us can sit by and say in smug unconcern 'This is not my affair.' While it is not so prevalent as gonorrhœa, it may beset us perhaps in disguise, and but too often in dangerous contagious form, in those unsuspecting hours when we believe ourselves at

ease among our friends. Syphilis is too cunning a craftsman in evil to permit the limitation of his labors to the few."

Locomotor ataxia, the slow creeping paralysis which affects the legs, the bladder, then the nerves of the eyes and stomach, is one of the horrible sequels of syphilis. General paresis, even more horrible, is also a late stage of syphilis. This disease converts a strong man into a helpless imbecile.

Gonorrhœa

The enormous prevalence of gonorrhœa and the appalling damage it inflicts make gonorrhœa a fitting twin for the other major scourge—syphilis.

Conservative estimates by Morrow and Forcheimer show that over 50 per cent of adult males have had gonorrhœa. Its difficulty of cure, and the fact that one attack produces no immunity, account in part for its amazing prevalence and the damage which must be charged to it.

The responsibility of gonorrhœa has been established for a large part of serious operations on both men and women, with invalidism and mortality usually charged under some other name.

Space does not permit an adequate discussion of these two diseases, but the essentials of their epidemiology and control, as given by the Committee of the American Public Health Association, are given below:

Syphilis.

1. *Infectious agent.—Treponema pallidum.*

2. *Source of infection.*—Discharges from the lesions of the skin and mucous membranes, and the blood of infected persons, and

articles freshly soiled with such discharges or blood in which the *Treponema pallidum* is present.

3. *Mode of transmission.*—By direct personal contact with infected persons, and indirectly by contact with discharges from lesions or with the blood of such persons.

4. *Incubation period.*—About three weeks. (In rare instances reported to have been as long as seventy days.)

5. *Period of communicability.*—As long as the lesions are open upon the skin or mucous membranes and until the body is freed from the infecting organisms, as shown by microscopic examination of material from ulcers and by serum reactions.

6. *Methods of control:*

(A) The infected individual and his environment—
  (1) Recognition of the disease—Clinical symptoms, confirmed by microscopical examination of discharges and by serum reactions.
  (2) Isolation—Exclusion from sexual contact and from preparation or serving of food during the early and active period of the disease; otherwise none, unless the patient is unwilling to heed, or is incapable of observing, the precautions required by the medical adviser.
  (3) Immunization—None.
  (4) Quarantine—None.
  (5) Concurrent disinfection of discharges and of articles soiled therewith.
  (6) Terminal disinfection—None.

(B) General measures—
  (1) Education in matters of sexual hygiene, particularly as to the fact that continence in both sexes and at all ages is compatible with health and development.
  (2) Provision for accurate and early diagnosis and treatment, in hospitals and dispensaries, of infected persons, with consideration for privacy of record, and provision for following cases until cured.

(3) Repression of prostitution by use of the police power and control of use of living premises.
(4) Restriction of sale of alcoholic beverages.
(5) Restriction of advertising of services or medicines for treatment of sex diseases, etc.
(6) Abandonment of the use of common towels, cups, and toilet articles and eating utensils.
(7) Exclusion of persons in the communicable stage of the disease from participation in the preparing and serving of food.
(8) Personal prophylaxis should be advised to those who expose themselves to opportunity to infection.

Gonorrhœa.

1. *Infectious agent:* Gonococcus.

2. *Source of infection:* Discharges from lesions of inflamed mucous membranes and glands of infected persons, *viz.* urethral, vaginal, cervical, conjunctival mucous membranes, and Bartholin's or Skene's glands in the female, and Cowper's and the prostate glands in the male.

3. *Mode of transmission:* By direct personal contact with infected persons, and indirectly by contact with articles freshly soiled with the discharges of such persons.

4. *Incubation period:* One to eight days, usually three to five days.

5. *Period of communicability:* As long as the gonococcus persists in any of the discharges, whether the infection be an old or a recent one.

6. *Methods of control:*

(A) The infected individual and his environment—
(1) Recognition of the disease—Clinical symptoms, confirmed by bacteriological examination or serum reaction.
(2) Isolation—When the lesions are in the genito-urinary tract, exclusion from sexual contact, and when the lesions are conjunctival, exclu-

sion from school or contact with children, as long as the discharges contain the infecting organism.

(3) Immunization—None.

(4) Quarantine—None.

(5) Concurrent disinfection—Discharges from lesions and articles soiled therewith.

(6) Terminal disinfection—None.

(B) General measures—

(1) Education in matters of sexual hygiene, particularly as to the fact that continence in both sexes at all ages is compatible with health and development.

(2) Provision for accurate and early diagnosis, and treatment in hospitals and dispensaries of infected persons with consideration for privacy of record and provision for following cases until cured.

(3) Repression of prostitution by use of police power and control of use of living premises.

(4) Restriction of sale of alcoholic beverages.

(5) Restrictions of advertising of services or medicines for the treatment of sex diseases, etc.

(6) Elimination of common towels and toilet articles from public places.

(7) Use of prophylactic silver solution in the eyes of the new born.

(8) Exclusion of persons in the communicable stage of the disease from participation in the preparing and serving of food.

(9) Personal prophylaxis should be advised to those who expose themselves to opportunity for infection.

CHAPTER XXXV

LEPROSY

Definition.—Leprosy is a slowly developing, chronic infectious disease, communicable only by intimate and prolonged contact. It is caused by the leprosy bacillus and is characterized by lesions of the skin and nerves. The leprous lesion of the skin or mucous membrane, or nodule, consists of a great multiplication of cells somewhat similar to the "tubercles" of tuberculosis, and the "gumma" in syphilis. The cell proliferation in the nodule is caused by the germs; the nodule later breaks down and ulcers result. The bacilli also attack the nerve cells of the peripheral nerves, spinal cord, and brain, causing varied symptoms from deranged nerve function. In some cases, the nodular type predominates; in others the smooth or anæsthetic type (nerve lesions); and in some cases both types of lesions (skin and nerve) are mixed.

History.—Leprosy is known to have existed before 1500 B.C. in the Delta of the Nile. The disease is described by Aristotle and other Greek writers, but not by Hippocrates, whose "lepra" was probably another disease—psoriasis. Leprosy is one of the oldest known diseases. That it existed long before the Christian era is certain, although some of the descriptions are open to dispute and may have included other diseases.

It was introduced into Greece from Persia during the

Persian wars, and the Roman conquerors of Greece presumably carried the disease to Italy. It spread all over Europe in the early centuries, and by the seventh century in France segregation of lepers was practiced. The disease increased so greatly in the Middle Ages that great numbers of lazarettos were established for the isolation of lepers.

Leprosy decreased in Europe from the fifteenth to the seventeenth century and became rare except in Russia, Finland, Norway, Sweden, and Denmark in the north, and Turkey, Greece, Italy, and Spain in the Mediterranean. There is at present considerable prevalence in Japan, China, India, and South Africa. It is common in the Philippine Islands and Hawaii. There is some leprosy in Porto Rico and the West Indies and Mexico, and in nearly all Central and South American countries.

Leprosy in the United States has several very distinct foci which apparently have a diverse origin for each. The leprosy on the Pacific Coast is Oriental in origin. The leprosy in Minnesota and the North Central States is closely connected with Scandinavian immigrants. The leprosy in Florida probably dates back to the days of the slave trade. The focus in East Texas may have a Mexican origin. The origin of the Louisiana focus is in dispute. It was claimed by many that it was introduced by the Acadians; but this is by no means certain, as it may have been introduced by Spanish or French from the West Indies.

While leprosy shows a liking for tropical and subtropical countries, it exists or has existed practically in all countries; and it is in the colder countries that it persists longest. When it disappeared from the warmer countries in Central Europe, it persisted in Norway, Finland, and Russia; and

it remained prevalent in North Scotland centuries after it had disappeared from England and Ireland.

Prevalence in the United States.—Leprosy is not a great problem in the United States, but there are many more lepers than the relatively few reported or isolated cases. A conservative estimate would place the lepers in the United States at about 1000. The federal government has purchased the Louisiana home for lepers at Carville, La., and lepers are being sent there from nearly every state in the Union. Because of the slow spread of the disease, state laws have been lax, and segregation was practiced only in California, Massachusetts, and Louisiana. Besides the largest focus in Louisiana, investigations of the Public Health Service show that the disease is endemic in Texas and Florida, occurring among natives who have never been out of the state.

The Cause.—The bacillus of leprosy, discovered by Hansen in 1874, is a slender rod-shaped organism. It is called an "acid fast" bacillus, because it retains the anilin dye (fuchsin) even after treatment with acids. In this and in other particulars, it resembles the tubercle bacillus. It is accepted as the cause of leprosy, because it is found in all cases of leprosy if a careful search is made.

There is a rat leprosy, which closely resembles human leprosy, caused by a germ apparently identical with human leprosy bacilli. It has been found in Australia, Japan, India, Paris, and California. It was not found in Hawaii, where human leprosy is common, and rat leprosy was found in Berlin where there is no human leprosy. This fact would indicate that rat leprosy has no relation to the spread of the disease in man.

Period of Incubation.—The period of incubation may

be very long in leprosy. There are well-authenticated cases where the disease developed years after the last or only possible exposure. Its communicability from person to person is accepted, but it has such a low infectivity that intimate close contact for a considerable period seems to be necessary to transmit the disease.

Immunity.—The very long incubation period, usually covering years, suggests that the human body must be very resistant to the infection of leprosy. Inoculations of human beings with leprous material have failed to produce leprosy, except in one doubtful case. This was a convict, who did develop leprosy, but the man had been in contact with leprous relatives before and after the inoculation. Man must have a relative immunity to leprosy, which is lowered or nullified by some of the conditions of transmission which are not understood.

Vaccines.—Clegg grew the bacillus by associating with it an ameba from dysenteric stools; this is a difficult and somewhat clumsy procedure. The fact that the bacillus does not grow satisfactorily outside the human body, makes it difficult to produce a vaccine. The most hopeful treatment has resulted from the use of derivatives of chaulmoogra oil.

Source and Mode of Transmission.—The source of infection is man, but the mode of transmission is not positively known. Attempts to produce the disease in animals have failed in every instance. Many insects have been accused of spreading leprosy; gnats, fleas, mites, lice, flies, and mosquitoes have fallen under suspicion; but transmission by these is unlikely when direct inoculation from man to man has been unsuccessful. It must be admitted that insect transmission is possible.

The frequency of nasal ulcers in leprosy leads many to

believe that the germs effect lodgment and enter the body through the nose. Leprosy bacilli may be found in the nose lesions when they are found nowhere else. While dogmatic assertion of the mode of transmission cannot be made, it is sufficient for practical purposes to know that intimate contact is essential to its spread. It is not hereditary, and children of leprous mothers are born free of the disease; although they may acquire the disease later from intimate contact with their parents.

Control.—Leprosy has been eradicated in England and other countries by segregation of lepers. The disease has persisted because segregation has not been carried out in all countries, or has been carried out in a very lax manner. The disease spreads so slowly in this country that there is excellent opportunity for isolating cases before they have infected others. Up to the present, we have not embraced this opportunity and leprosy has probably increased in certain areas.

We have now a Federal Home for Lepers, which serves a twofold purpose: It gives humane care and excellent medical treatment to the individual leper, and at the same time removes him from the community where he would probably cause other cases. The Federal Home for Lepers is managed on such lines that patients stay without restraint. It is not a prison, but the most modern hospital or sanitarium colony of the cottage type. Another reason why lepers compete to gain entrance and need no restraint to hold them, is the specific treatment developed by the Public Health Service. This is the administration of derivatives of chaulmoogra oil, which seems to be curative. At least, arrested cases in Hawaii have been paroled under observation, and it is hoped that these seeming cures may be permanent.

CHAPTER XXXVI

TRACHOMA AND FAVUS

Trachoma

Definition.—Trachoma is a chronic eye disease, with destructive inflammation of the conjunctiva. It is contagious and without treatment causes great damage to the eye and often results in blindness. It has been known for centuries and very common since ancient times in Egypt, Levantine countries, and Southern Europe. It became so common in our hospitals in seaport cities that, in 1897, the American Ophthalmological Society urged the United States Government to exclude incoming aliens with trachoma. This has been done since 1897. Thousands of cases are excluded each year at our seaports, and the writer, while on duty at Naples, Italy, rejected over seven thousand cases in a year among immigrants bound for the United States.

Since 1912, the Public Health Service has demonstrated not only that there was a considerable prevalence in the United States, but that much of this was probably not traceable to foreign immigration. McMullen demonstrated its prevalence in mountain sections of Kentucky, which had no foreign immigration and very little contact with the outside world.

Within three years (1912-1915) the U. S. Public Health Service examined 192,000 persons, mostly school children,

in sixteen states, and found over 3600 infected. During the same investigation service officers found that out of 39,000 Indians in twenty-four states, nearly 9000 were infected, or over 22 per cent of all examined. Trachoma has become, therefore, a serious problem in many parts of the United States. It is a disease of insanitary housing conditions, filth and overcrowding.

Cause.—The cause of trachoma is in dispute and must be set down as unknown. Prowaczek, in 1907, described the so-called trachoma bodies; these resemble protozoan parasites and are believed by some to be such, and the cause of the disease. Others regard them as products of cell destruction and their presence as incidental not causative. The Koch-Weeks bacillus is believed by some to cause trachoma. While the specific cause is in doubt, there is no doubt whatever of its contagiousness.

Source.—The source of trachoma is always another human case; the virus is contained in the discharges from the eyes.

Mode of Transmission.—The virus is transmitted by direct contact with infected persons, or indirectly by means of towels or articles recently soiled with the eye discharges of a trachoma case. A case of trachoma must be regarded as infectious until all inflammatory tissue has disappeared. The process usually goes on through ulceration until the inflamed tissue is replaced by scar tissue.

Control.—There are two parts to our control program: keeping out additional cases of trachoma, and taking care of what we already have. The foreign importation is now almost negligible, because of our rigid inspection at foreign and United States seaports, and the deportation of aliens found suffering with the disease. A fine of $100 also acts

as a deterrent on steamship companies from bringing in infected aliens.

The attack on the disease in American communities must be aimed at not only the disease in the individual, but at his insanitary environment as well. The best results are obtained by educational methods among the school children, and surgical treatment of the individuals infected.

Trachoma was regarded for years as an incurable disease, but McMullen, of the U. S. Public Health Service, devised a surgical procedure which cures in a large proportion of cases. McMullen has operated small hospitals in sections of Kentucky and other states, in which surgical treatment cleared up the existing trachoma; and at the same time, these little hospitals served as educational centers from which personal and family hygiene was taught and spread over a wide area. The work initiated by McMullen is being continued by Kentucky and other states.

Favus

Definition.—Favus is a skin disease, with special predilection for the hair and nails, characterized by the formation of yellow crusts, destruction of the hair follicles, and permanent loss of hair.

Cause.—It is caused by a fungus known as the *achorion schoenleinii.*

Source.—Other human cases of favus, also dogs, cats, mice, rabbits, and fowls. It is a filth disease and thrives in overcrowded, squalid habitations and institutions.

It is usually transmitted by direct contact, indirectly through toilet articles. It is communicable until skin and

scalp lesions are healed, and persistent intensive treatment is required for cure. Favus is more common in Europe than America, and it is so common in immigrants that it is a mandatory cause of exclusion under our immigration laws.